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PERITHELIOMA OF UPPER EYELID . . . N. R. DONNELL

AN EXPERIMENTAL STUDY OF OPHTHALMIA IN RATS ON RATIONS DEFICIENT IN VITAMIN A . ARTHUR M. YUDKIN

A NEW THEORY OF ACCOMMODATION . LINDSAY JOHNSON

A FACTOR IN INFECTION AFTER NEEDLING . RALPH I. LLOYD

TREATMENT OF TRACHOMA WITH STRONG SOLUTIONS OF SILVER NITRATE . . . . . N. R. DONNELL

IRIDO-CYCLITIS SYMPATHICA FOLLOWING AN IRIDECTOMY FOR GLAUCOMA SECONDARY TO SARCOMA OF THE CHOROID C. W. TOOKER AND H. D. LAMB

REPORT OF A CASE OF CHLOROMA OF THE ORBIT CYRIL BARNERT

AUTOPSY REPORT OF A CASE OF BILATERAL GLIOMA RETINÆ WITH GLIOMATOUS INVOLVEMENT OF THE IRIS ARNOLD KNAPP

THE NEW UNIT OF VISUAL ACUITY AND ITS PRACTICAL USE L. DE BLASKOVICS

(Complete contents inside)

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ARCHIVES OF OPHTHALMOLOGY.

PERITHELIOMA OF UPPER EYELID.[1]

BY DR. N. R. DONNELL, ST. LOUIS.

(With one illustration on Text-Plate XVII.)

MRS. M. J. O., age 74, applied for treatment April 5, 1923, because of a growth of left eye.

History previous to present trouble of no interest except that she consulted me in the summer of 1917 for a chalazion of the left upper eyelid, which presented nothing unusual in appearance and which was incised and curetted and so far as I know made the usual recovery. I have no record of this visit; the above statements were elicited from the history and from my own recollection of the case.

Present trouble began about five years previous to my seeing her one year ago. There appeared first, a small lump under the left upper lid. Thinking it similar to the previous trouble she gave it little attention as it gave her no inconvenience until the summer of 1922. At that time she received a fall, bruising the left side of her head and face, after which the growth became active and grew very rapidly. She still did not seek advice till she came to me in April, 1923, at which time the findings were as follows:

V.O.D. $-\frac{20}{100}$. V.O.S.—counting fingers at 3 feet.

My inability to get this large tumor mass out of the line of sight was partly responsible for her lowered vision. This growth was about the size of a walnut extending well out between the lids, making closure of the lids impossible and a complete examination of the globe difficult. It appeared to have its origin from well back under the upper lid. The outer coats of the globe in the region of the tumor were dis-

[1] Read before the St. Louis Ophthalmic Society, March 28, 1924.

colored, showing very dark. The media appeared clear, but the exact condition could not be made out. The tumor seemed lobulated and in places almost cystic as though some portions had broken down. Patient had had no pain, the only inconvenience being the mechanical irritation and the tendency to bleed if manipulated.

This case was seen by Dr. F. E. Woodruff and Dr. H. D. Lamb who felt as I did, that there was little doubt of the malignancy of its nature and that the immediate removal was necessary.

Being unable to determine the full extent of this growth it was thought best to consult a general surgeon, whereupon Dr. Wm. E. Leighton was asked to see the case. He likewise advised the early and complete removal of the growth.

The patient was sent to the hospital and on April 15, 1923, was placed under a general anesthetic and a section of the tumor removed and examined by Dr. Ralph L. Thompson who brought back a report from an examination of the frozen section of an angiosarcoma. The question of course was whether to remove only the tumor or to do a complete evisceration of the orbit; after this report the latter course was followed. There appeared to be a good deal of discoloration of the upper lid but since there seemed to be no actual invasion we concluded the discoloration was due to circulatory disturbance and would recover. As the operation proceeded the growth seemed to have its origin in the tarsus—doubtless from the old chalazion removed in 1917—and when it was removed the whole tarsus appeared to have been absorbed in the growth.

The patient made an uneventful recovery and up to three months ago had shown no evidence of a recurrence.

Dr. Thompson's subsequent report was angiosarcoma with perithelioma appearance in places.

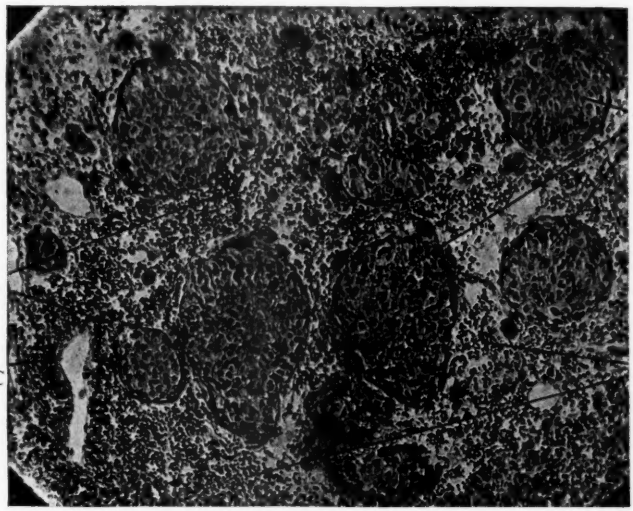
Dr. D. H. Dolley pronounced the tumor a typical perithelioma.

PATHOLOGICAL EXAMINATION OF THE TUMOR BY DR. H. D. LAMB.

Because of its great friability, the tumor was broken into fragments in its removal and the location of these portions in vivo was lost. In the fresh state immediately after excision, it was noticeable that the pieces were quite soft, had very shaggy fracture-surfaces and showed much hemorrhage.

Portions of the growth were fixed in 10% formalin, imbedded in paraffin and stained with hemotoxylin and eosin.

ILLUSTRATING DR. N. R. DONNELL'S ARTICLE ON "PERITHELIOMA OF UPPER EYELID."



Typical section through perithelioma of upper eyelid, showing groups of tumor cells (T), red corpuscles (R), small lymphocytes (L), cystic space (C).

Macroscopically the stained sections showed that the growth was of a fairly dense structure with considerable hemorrhage.

Microscopic examination revealed the composition of the tissue for the most part to be solid cords or masses of cells, these cellular groups being of greatly varying size and shape. Between these cell-masses there are present red blood cells in compact arrangement. Such an appearance is somewhat similar to that of glioma retinæ. Small lymphocytes are seen among the red corpuscles, for the most part diffusely scattered but here and there appearing in fairly dense groups. Also among the red blood cells there are seen here and there empty cystic spaces of varying size and shape, lined with a thin layer of flat endothelium; these cystic spaces are found in the greatest quantity where there are wider masses of red blood cells, that is where the essential cellular masses are few and far between. In some portions of the growth, however, there is no alveolar arrangement of the cells but the appearance is that of sarcoma with large areas consisting of only cells and blood vessels.

In the midst of many of the cords or masses of cells is seen a thin walled blood vessel, although such vessel does not always lie centrally as in the case of the cellular groups of glioma retinæ. The changes to the essential cells leading to the somewhat similar typical picture in perithelioma and retinæ are quite similar, the cells farthest from the blood glioma vessels atrophy and necrose, because of lack of nutrition and are absorbed; the latter process has proceeded further in the case of our perithelioma specimen than in the usual glioma picture, for no necrotic cells are present in our sections between the solid cords and masses of essential cells.

The essential cells are found to be generally large although they vary considerably in size due to the cells in some places being much more crowded together than in others; they are round or oval shape when they occur loosely arranged but polyhedral, when crowded together as is usually the case. There is practically no intercellular substance; a few fine connective tissue fibrils are seen here and there between the cells. The nuclei of these cells are quite large both absolutely and in relation to the cytoplasm although their size also varies greatly. The nuclei take the stain faintly and well marked nucleoli can usually be seen. The cytoplasm also stains faintly and in many places is vacuolated and in other places represented by a group of fine granules.

Quite commonly seen are very large or giant cells although they would be called small giant cells. These show either a single very large and oval nucleus, two or more nuclei or

a large multilobed nucleus. The nuclei in these giant cells usually stain quite darkly with hemotoxylin.

Where the cells are not present in alveolar arrangement but in extensive cellular masses, the size of the individual cells is more uniform and there are fewer giant cells.

In some portions of the growth in the cell groups and masses there is a small to moderate amount of diffuse infiltration with small lymphocytes, polymorphonuclear leucocytes or combinations of these two kinds of cells in varying proportions.

Perithelioma according to Ewing in his work entitled *Neoplastic Diseases* is a term applicable to a definite group of tumors, but the exact origin of these tumors and their relation to other neoplasms are questions requiring more extensive data than we now possess. Eberth in 1870 published in Virchow's *Archives* the statement that the blood-vessels of the brain and spinal cord with the exception of the capillaries are covered by an external epithelium, for which he thought the term "perithelium" would be most appropriate. Ewing states that although Eberth did not clearly separate these perithelial cells from the endothelium of peri-vascular lymphatics, similar cells have been described by others about the blood-vessels of the hypophysis, carotid gland, adrenal, breast, salivary gland and testis. Nevertheless the existence of such cells has not been accepted as a well established fact in normal histology, and the derivation of an important group of tumors rests on an uncertain basis. The belief that certain tumors do arise from Eberth's perithelial cells is suggested chiefly by the peculiar arrangement of the tumor-cells, radiating out from the supporting vessels, but no such tumor has yet been clearly traced to perithelial cells. Accordingly many other terms have been employed by different authors for this type of growth—as angiosarcoma, plexiform angiosarcoma, perivascular endothelioma, and periendothelioma.

Gross Appearance.—According to Ewing, perithelioma usually appears as a well circumscribed very vascular, at first slowly, later rapidly growing tumor, which may reach considerable dimensions. It is very subject to hemorrhage, necrosis and ulceration. In early stages it may be freely movable in the tissues exciting little local reaction, and may be firm, soft or almost fluctuating or pulsating. Local extensions and metastases occur late, but after imperfect removal recurrence, rapid extension, metastases and cachexia supervene, so that the general prognosis is unfavorable.

Structure.—The typical perithelioma presents a very characteristic structure. It is composed of congeries of

small vessels of the type of arterioles sheathed with large polyhedral, fusiform or rounded cells, which radiate in layers from the vessel wall. At a certain distance from the vessels nutrition often fails and the cells undergo anemic necrosis which serves to isolate and emphasize the vascular units. In many tumors the vessels disappear, the growth is of diffuse large spindle-cells and the tumor resembles simple sarcoma. Giant-cells of small and large dimensions with multilobed nuclei are a frequent and characteristic feature especially in the diffuse growths.

Clinical Types.—The subcutaneous tissue is one of the chief seats of typical perithelioma. The loose tissues of the anal region, orbit, axilla, popliteal space and the regions of embryonal fissures but not the more exposed regions, furnish the exact points of origin and this distribution suggests a relation to embryonal disturbance of superfluous tissue. Other regions to be affected by this type of growth are the deep fascial and intermuscular septa, the buccal mucosa over the hard and soft palate, the pia mater, the bones, breast, kidney and carotid gland.

Under the term "perithelioma" there could be found in the literature but three cases of this form of neoplasm involving the eyelid. One reported by J. Eicke (*Klin. Mon. f. Aug.*, 51, 1, May, 1913, p. 588) was removed from the skin of the upper eyelid. A second reported by R. S. Lamb (*Trans. Am. Acad. Ophth. and Oto-Laryn.*, 1912, page 111) occurred on the bulbar conjunctiva apparently secondary to injury; after removal it recurred on the eyeball and both eyelids. A third was reported by R. S. Charsley (*Amer. Journ. Ophth.*, v. 3, 1920, p. 687), who removed a growth from the eyelid which had been incised as a probable chalazion. Two months later the temporal bone was involved and after six months, death occurred, with signs of metastasis into the spine.

AN EXPERIMENTAL STUDY OF OPHTHALMIA IN RATS ON RATIONS DEFICIENT IN VITAMIN A.[1]

By Dr. ARTHUR M. YUDKIN, New Haven.

THE early recognition of the relative importance and the possible relationship of certain ocular disease in experimental animals to the lack of newly established factors in nutrition belongs to the biochemists. Thus, long before the vitamin hypothesis was formulated, Falta and Noeggerath (1) observed that rats fed on a food mixture of casein, lard, starch, dextrose and inorganic salts would not grow and maintain life. They found that these animals developed an eye disease. Paul Knapp (2) reinvestigated this experiment and found that there was some relationship between the eye disease and the nutritional disorder.

In 1913 Osborne and Mendel (3) noticed that animals on food mixtures alike in all other respects, would continue to grow and thrive or would stop growing and shortly thereafter die, according to whether the fat in the diet was butter fat or lard. At this time they recorded the fact that animals suffering from the lack of the indispensable fat soluble substance were subject to an ophthalmia. Later McCollum and Davis (4), working along the same lines, also made this observation. Even though the earlier workers did not appreciate the connection between the eye disease and the dietary factor,

[1] This investigation was made in co-operation with Drs. Thomas B. Osborne and Lafayette B. Mendel, under whose direction the feeding of the experimental animals was conducted. The histological studies were conducted in the Department of Pathology and Bacteriology, Yale University School of Medicine. The expenses of the research were defrayed in part by the Carnegie Institution of Washington, Washington, D. C.

a sufficient number of investigators in various places observed this ocular phenomenon and its apparent relation to the lack of vitamin A in the diet, so that today we know that it is more than a chance coincidence.

The eye lesions developing in animals as a result of the lack of vitamin A have been variously named; ophthalmia, keratomalacia, xerophthalmia and xerosis corneæ. The different names given to the abnormal ocular condition are derived from the nomenclature of the eye disease of man. The present study was taken up in order to show the possible resemblance to that of the human eye disturbance similarly labeled.

With the view of examining the progressive ocular changes from a clinical and pathological standpoint, a series of six groups were studied. The first, known as the control group, consisted of animals fed a mixed diet. The second group, consisted of animals fed a mixture lacking vitamin A, was killed when they showed excessive lachrymation and beginning photophobia with a slight suggestion of sanguinous tinging of the secretion. The third group of animals, fed on the same diet as the second, was allowed to progress to the stage at which plaques, fatty in appearance, accumulated in the fornices and the cornea showed some signs of dryness and loss of corneal reflex. The fourth group of animals was continued to the stage at which the cornea showed signs of breaking down. The fifth group were fed on the same diet except that another element was lacking. The sixth group consisted of animals fed on the diet void of vitamin A until the ocular disturbance was well defined, and their nutritional decline was such that if allowed to go on would die, before cod liver oil or butter fat was added to the diet to secure recovery.

In order to determine the character and site of origin of the initial lesion and the sequence of changes leading up to the condition of xerosis conjunctiva and cornea and subsequently terminating with keratomalacia as described in a previous paper (5), a careful histological study was made of the eye and the adjacent structures, including the lids and paraocular glands.

Young white rats, free from any visible disease, varying in age from 21–30 days, and weighing from 45–50 grams were placed on the following diet which was lacking vitamin A.

Casein (nearly pure)	18 gm.
Artificial Salts IV	4 gm.
Starch	54 gm.
Lard	24 gm.
Yeast	0.20 gm.

Instead of following the typical growth curve there was a falling off from the normal and simultaneously with this phenomenon an ocular disturbance appeared, which symptom developed after varying periods of from 30–60 days. The animals were sacrificed at the different stages of the ocular disturbance in order to study the progressive changes histologically. To make sure that anesthesia did not cause excessive secretion of the paraocular glands and did not injure the conjunctiva of the lids and cornea, the animals were first stunned and then guillotined. The whole head was placed in either Zenker's fluid or formalin 10%. After proper fixation the extra obital lachrymal gland was dissected from in front of the ear, together with its duct leading to the intraorbital lachrymal gland at the outer angle of the orbit. The eyelids with a margin of skin were left attached to the eye, as was also the Harderian gland, which almost completely surrounds the eyeball. The lens was removed through a window made in the sclera before the whole tissue was imbedded in paraffin.

While there is no sharp demarcation between group two and three, and three and four, a brief clinical picture will make clear the basis of division. The first sign of ocular disturbance is an increase of lachrymation accompanied by photophobia. Then the eye, normally prominent and protruding, seems to sink into its bony socket and the animal appears to dread the light more and more. The lachrymal secretion changes from a watery to a viscid, sanguinous tinged secretion. A slight edema of the eyelids becomes manifest and there is an accumulation of a dry secretion in the ocular cul-de-sac. The lid hairs fall out and the lids become matted together. In the early stages, the cornea shows no visible changes except for the marked congestion about its junction with the palpebral conjunctiva. With the progress of the eye disturbance, the cornea appears hazy particularly about the periphery. Patches of secretion which are now fat-like, and yellowish in appearance extend on to the corneal surface from the fornices;

frequently they are seen on the center of the cornea. The cornea becomes dry, oily and lusterless, losing its normal transparency. When the plaques are removed from the cornea, the surface does not show any disturbance and it will not stain with fluorescein. The ocular lesion will go on to corneal ulceration, perforation and panophthalmitis, if the animal does not succumb. It is these latter stages that have been studied and described by other investigators (6).

Before presenting the histological findings, a brief discussion of the anatomy of the cornea and the paraocular glands together with their physiology may make for clearness. The structure of the cornea varies very little from that of the human tissue. The lachrymal apparatus consists of two parts; one the extraorbital portion lying in front of the ear and above the parotid gland and the other intraorbital gland lying at the outer angle of the orbit. The former is connected to the latter by a small duct. They secrete their watery fluid into the cul-de-sac at its outer canthus. In addition the eye is lubricated by a more viscid secretion supplied by the Harderian gland situated intraorbitally and in close apposition to the eyeball and secreting its fluid at the inner canthus between the lid and the nictitating membrane. This gland is quite large as has been pointed out in a previous paper (7).

While both glands are acinotubular in type and are similarly lobulated, their component cells are different. The lachrymal gland is made up of small acini which, at least in fixed preparation, appear to have little or no lumen. The nuclei vary markedly in size and are rich in chromatin. The cytoplasm takes a definitely basophilic stain. Large vacuoles are numerous. In the Harderian gland the acini are larger and the cells are uniform in size with relatively small nuclei and abundant eosin-staining cytoplasm.

The animals of group one together with about six old rats suffering from chronic skin and eye disease, served as controls. To this was added some animals fed on the same diet as group five except for the addition of vitamin A.

Animals of group two which showed the earliest ocular disturbance clinically, also presented small focal inflammatory lesions in the lid and the nictitating membrane. These consisted of an infiltration of the epithelial layer by polynuclear

and mononuclear leucocytes, with degenerative changes in the epithelium. In places there was keratinization of the superficial layers of the epithelium with an accumulation in the conjunctival sac of keratin shreds, polymorphonuclear leucocytes, red blood cells and cellular debris. In respect to the location of the lesion, however, some variation was observed. The site of election is in the lid near the palpebral fold. In some instances, foci were seen on the nictitating membrane. Sections through the eye and lids, at different levels, indicate that several foci may be present. Both the upper and lower lids are frequently simultaneously involved; that is, the changes appear equally advanced. The cornea appears unaltered as far as can be seen. The intra-and extra-orbital glands show nothing abnormal. The Harderian gland, however, appears to have a definite granulation and vacuolization of the cytoplasm. The distortion of the cells is really pathological. I did, however, notice this change in two supposedly normal Harderian glands. It is quite possible that this change represents a functional activity. Only slight deviations from this picture were found in this whole group.

The third group of animals may be classed as moderately advanced. The proliferation of the epithelium, with slight keratosis at points just opposite the early lid lesions, appears to be the first change in the cornea. The inner layer of epithelial cells lose their mosaic pattern-like regularity in places and there is an invasion of leucocytic or mononuclear cells. A lateral ingrowth of blood vessels from the limbus into the substantia propria immediately follows, and in fulminating cases, only a few days elapse before the cornea is markedly edematous. The tissue is permeated with budding capillaries and proliferating corneal corpuscles, and infiltrated by wandering cells, chiefly of polymorphonuclear type. In more slowly developing lesions, mononuclear cells may predominate. The corneal epithelium and the substantia propria shows marked swelling particularly at the limbus. Considerable thickening of the former with keratinization and a shelling off of the keratinized epidermis is very regularly seen. Bowman's membrane, Descemet's membrane and the posterior endothelium are found to be intact.

The lid changes in this group show very little progress from those already noted. There is generally no extension of the infection to the deeper tissues of the lid. Edema of the lids, noted clinically, is manifest in the microscopic section. The nictitating membrane exhibits alterations similar to those of the cornea—edema and cellular infiltration in the inner epithelial and connective tissue layers.

The Harderian gland grossly shows marked diminution in size in comparison to the control animals. Lesions of the Harderian gland were found in this group, but the incidence of involvement was highest in rats showing advanced conjunctival and corneal changes. There is no sharp line of division between group three and four, since the one passes over into the other. In describing the transformations that take place in this gland, it would be best to point out the definite changes that we find in the moderately advanced and advanced stages, rather than to try to separate them.

Even though granulation and vacuolization of cytoplasm was seen in some of the normal control specimens, this was a constant occurrence in the experimental groups. The advanced ophthalmia almost constantly showed extreme vacuolization accompanied by an apparent disintegration of the cytoplasm. In one gland, widespread necrosis of the epithelium was seen with only a slight apparently beginning, inflammatory reaction: a lesion which Lambert thought was comparable to the necrosis of the renal epithelium in mercury or uranium nitrate poisoning. In some a marked dilatation of the acini constitutes the most striking alteration. In some of the glands studied an acute inflammatory reaction was found. The histological features of the reaction did not differ essentially from those seen in other glandular organs. The acini and the interacinal tissue were often filled and infiltered with polynuclear leucocytes. In some instances, the lesions were focal, involving only one or a small group of lobules; but in many cases the entire gland was involved. The ducts were markedly dilated and the lumen was filled with polymorphonuclear leucocytes and cellular debris. The acute process generally passes over into a chronic inflammation with mononuclear cell infiltration, fibrosis, and atrophy. There was frequently seen what might be termed a combination of

acute and chronic inflammation; that is, the process was quite active, with numerous polymorphonuclear leucocytes, but associated with this was evidence of healing, with granulation tissue and well developed scar tissue, and numerous phagocytic mononuclear cells. Definite suppuration was rarely found.

The histological examination of the intra and extra-lachrymal gland showed no definite changes in the structure, other than that observed in the normal gland. It is possible that special granule stain may be necessary to demonstrate the change.

The fourth group represents the advanced stages of ophthalmic disorder and nutritional decline. The corneal epithelium shows a diffuse inflammatory reaction and the changes are similar to those seen in group three except more aggravated. A feature of the advanced lesion, is the remarkable vitality of the cornea seen in most cases. Occasionally ulceration develops rapidly, and in the majority of the cases, ulcers will appear sooner or later. But in any case, the necrotizing process represents a terminal stage.

The anterior chamber in some cases shows an accumulation of fibrin, mononuclear cells and pus. The iris is involved by the inflammatory process and shows considerable edema, cellular invasion and vascular engorgment.

In some cases there is definite breaking down of Bowman's membrane and infiltration of the stroma with mononuclear and polynuclear cells. The epithelium of the cornea invades the necrosed area with marked proliferation and numerous mitoses. Very few definite breaks in Descemet's membrane are observed.

Edema of the lids is evident. Occasionally an infection of the skin, similar to that in the conjunctiva, is also observed. The large lymphatic channels within the fibromuscular ring which supports the hair are often filled with considerable hemorrhage. The Meibomian ducts are conspicuously large but similar pictures were noted in the control animals and were therefore not considered as a pathological phenomenon.

Besides the lack of vitamin A, the diet of group five was deficient in a second factor such as phosphorus. The ocular manifestations appeared much sooner in these animals, and

were very severe from the start. Histologically the cornea showed a picture of fulminating suppurative keratitis. The superficial epithelial cells are swollen and in places there is a vacuolization and rupture of the upper layers causing unevenness and marked desquamation. Ulceration is present in some of the sections with extensive polymorphonuclear invasion and edema. The conjunctival epithelium of the lid and nictitating membrane also show an acute inflammation accompanied by considerable edema. The Harderian gland shows active functional disturbance and resembles the picture seen in the advanced stages of xerosis.

The last group of animals were treated with vitamin A to procure their recovery. The cornea had regenerated itself almost completely. In one case, vacuolization of the basal epithelium leaving free nuclei appeared. Another case showed the end result of deep ulceration, *i.e.*, ill formed layers of epithelium cells displacing the broken Bowman membrane and a cellular and vascular invasion of the substantia propria. In many of the sections remains of vascularization without any other pathological changes were seen. The Harderian gland almost completely reverted to its normal status when treated. There were, however, foci of degeneration seen in some of the lobes. Considerable granulation and vacuolization of the cytoplasm was observed and the tissue appeared to be more vascular. But the general finding was complete restoration of the ocular tissue to the normal state.

This work confirms the findings of many observers (8) that an ocular disturbance may be brought about by the lack of vitamin in the food. The initial lesion is seen in the conjunctiva of the lid and nictitating membrane. The process is a progressive one and the destruction of the cornea, which constitutes the most conspicuous picture of this ocular manifestation, is a secondary phenomenon. Pathologically this disturbance resembles the condition known as xerosis conjunctivæ and cornea and keratomalacia described in Graefe-Saemisch, *Handbuch der Augenheilkunde* (9). Clinically it resembles the eye disease described as Hikan (10) in Japan, Brazilian Ophthalmia (11) in South America, and Xerophthalmia (12) seen more recently in Sweden and Denmark. The fact that, as a general rule, both eyes are simultaneously

involved and that the progress in both proceeds at about the same pace, is very suggestive of some systemic disturbance.

Cramer (13) believes that the lack of vitamin A causes a two-fold lesion, a lesion in the intestinal mucosa and a diminution in the number of platelets and hence the normal mechanism of defence against bacterial invasion is broken down. Smith and Wason (14) have shown that in animals fed on a diet deficient in vitamin, the bacterial titer of the serum is distinctly lowered and that there is a less marked reduction in the bacteriatropic activity of the serum. This may be due to a deficient production of anti-bodies or a diminished amount of platelets in the blood. It is my opinion that the eye condition is primarily due to altered secretion of the paraglands which I have described.

SUMMARY.

The experimental ocular disturbance in the albino rat is due to the lack of vitamin A in the diet and if a second element is missing, the condition is more fulminating. It seems as though the protective substances in the secretions of the paraocular glands are so diminished as a result of the insufficient diet that the avirulent bacteria constantly found in the cul-de-sac take advantage of the situation and a pathological picture of a progressive low grade infection is present. The rapidity of the development and the degree of destruction probably depend in large part on the normal storage of the missing vitamin, the type of bacterial infection, the breaking down of the normal mechanism of the intestinal tract and the immunity of the body against secondary infection because of the diminution of the platelets and anti-bodies or their likes in the blood stream.

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A NEW THEORY OF ACCOMMODATION.

By Dr. LINDSAY JOHNSON, Durban, So. Africa.

IT is well known that we owe our present theory of accommodation to the researches of Helmholtz. You will find it described with slight variations in every text-book of physiology in existence. It is briefly as follows:

The crystalline lens is a biconvex highly elastic body, more convex behind than in front. It is surrounded by a capsule of fibrous tissue said to be highly elastic, which is considerably thicker in front. The lens is held in position by the suspensory ligament, which extends from the circumference of the lens and its capsule to the lining membrane covering the ciliary processes, which latter are continuous with the choroid behind and the iris in front. The ciliary muscle consists of unstriped muscular fibers which form a circular band about three *mm* broad, which encircles the eyeball between the sclerotic and the ciliary processes. These fibers spread out like a fan from the base of the iris, spreading out into the meshes of the choroid and the delicate fibers in the supra-choroidal space. In addition to this muscle, there is a second unstriped circular muscle which is placed between the ciliary fibers and the ciliary processes. The fibers of this muscle, which go by the name of the fibers of Müller, run at right angles to the former muscle, and surround the margin of the lens. To the best of my knowledge Helmholtz omits mention these fibers altogether in connection with his theory, as they have no bearing on it. He accounts for accommodation as follows:

When the eye accommodates so as to bring the point of fixation nearer the eye, the choroid is drawn forwards by the

action of the radiating fibers of the ciliary muscle; and the ciliary processes in consequence are squeezed or pushed forwards towards the circumference of the lens. Since the suspensory ligament is attached to the tips of the ciliary processes on the one hand, and to the capsule of the lens on the other, the capsule, being highly elastic, becomes relaxed, and allows the anterior surface of the lens to bulge forwards, which increases its convexity, and consequently its refractive power. At the same time the ciliary processes being continuous with the iris, pushes the latter forwards, and thus contracts the pupil. When accommodation is at rest, the lens capsule is put on the stretch, and flattens the front surface of the lens, whereby the refractive power of the lens is diminished. This as far as I can gather is Helmholtz' theory.

Now I should like to point out that because a man has attained a great reputation by reason of his remarkable achievements and ability we have no right to assume that everything he says must therefore be accepted as true. This is especially the case in regard to hypotheses and theories. Helmholtz, like many other great men, at times invented theories to account for certain phenomena which when examined critically would not hold water for a moment. Because we do not happen to possess the ability of Helmholtz, it does not follow that we are not equally capable of discovering new facts or of hitting on the true explanation of a phenomenon. The late Lord Kelvin made some hypotheses which had I made them I should have been laughed out of court. For instance he accounted for the origin of life on this planet by suggesting that the germs which were the progenitors of every living thing on our earth including ourselves, came traveling here on the back of a meteorite! while Newton who was probably the greatest genius the world has ever produced, because he happened to examine certain pieces of glass in which the refractive indices bore a constant proportion to the dispersion, came to the erroneous conclusion that it was impossible to construct an achromatic object-glass, a mistake which delayed the construction of optical instruments for nearly a hundred and fifty years, in fact it was not until the optician Dolland, doubting the correctness of Newton's statement, re-investigated the matter and constructed an achromatic telescope

that the error was rectified. I have digressed somewhat from the subject in order that our readers may be inclined to give me credit for a little intelligence, and give my theory some consideration, before instantly dismissing it because it happens to run contrary to Helmholtz' theory.

Now in the first place Helmholtz omits to mention the circular fibers of Müller as taking part in the act of accommodation. They are of the highest importance because by their contraction they greatly assist in pulling inwards (*i.e.* towards the margin of the lens) the ciliary processes. I have made repeated examinations of the ciliary processes during accommodation, but I have never been able to satisfy myself that they actually touch the capsule. But we know that these fibers are more or less atrophied in myopic eyes, since myopes never have to use their accommodation except for regarding objects near at hand.

Then Helmholtz tells us that when the ciliary muscle contracts, it relaxes the suspensory ligament and with it the capsule and so allows the lens (which he says is normally compressed and flattened by the tension on the capsule) to bulge forwards, and so increase its curvature in front, which raises the refractive power of the eye. Now there is not the slightest proof that this takes place. I have spent very many hours in examining the eyes of human beings and various animals, but have never observed the slightest indication of this happening. In fact I fail to see how it could happen. The capsule is firmly attached to the lens around its margin and when the ciliary processes bulge forward they merely pucker up the suspensory ligament into folds, but do not affect the capsule in the least. I fail to see how they can, seeing that the capsule is attached to the margin of the lens all the way round. In order to prove whether this was correct I took out the eye of a rabbit, a guinea-pig, and an ox, and repeated the experiment on all three in the following way. I opened the eye with a razor and carefully extracted the lens without in any way injuring it. As I found it impossible to preserve the ligament intact, as it always tears away and is lost, I made an artificial ligament by placing the lens between two pieces of the thin vitelline membrane which lines the shell of a hen's egg. Then I applied a few touches of melted beeswax and

rosin (Prout's elastic glue will do just as well if not better) with the pointed end of a match just outside the edge of the lens in a few places, so as to keep the two layers of membrane in contact without attaching them to the lens itself.

Now I found it impossible do what I would to effect any change in the curvature of the lens, no matter how I manipulated the artificial suspensory ligament. I trimmed it to the correct size, viz., three millimeters all round the edge of the lens, but it made no difference. I could neither flatten nor bulge it.

Again Helmholtz tells us that the capsule is kept on the stretch when accommodation is relaxed and *compresses the lens in front!* Surely this continual compression must fatigue the ciliary and indirectly the eye and cause pain or eyestrain. I have tried everything I could think of to flatten the lens but have never succeeded in doing so. I then made the following crucial experiment. I took a rabbit and passed a broad needle through the cornea. Then I dipped a capsule needle into some thick prussian-blue paint, and passing it carefully through the opening I made a clean vertical cut through the capsule and then examined the stained edges of the wound with the ophthalmoscope. Now if Helmholtz' theory were correct, the edges should have retracted, since according to him the capsule is highly elastic and kept on the stretch and only relaxed during accommodation. But nothing of the sort took place. The wound did not gape. This clearly showed that the ciliary muscle cannot cause the capsule to relax and so allow the lens to bulge forwards.

Between the suspensory ligament and the hyaloid membrane covering the vitreous is a tiny triangular space, sometimes called the Canal of Petit. This is bounded internally by the capsule of the lens, externally by the ciliary processes, behind by the hyaloid and vitreous, and in front by the suspensory ligament. Now what happens during the act of accommodation is as follows:

The two ciliary muscles (the radiating and the circular muscles) contract and their combined action drags forward the choroid and at the same time squeezes up the ciliary processes and pushes them into the circumlental space with the help of the circular muscle of Müller. This tends to compress the fluid in this space. But fluids are incompressi-

ble, so something has to give way. This can only be the capsule which is forced inwards compressing the lens round its circumference, and thus causing the two surfaces to bulge. Now the posterior surface cannot be displaced backwards because it rests against the incompressible vitreous. So it must bulge forwards. But here it presses against the equally incompressible aqueous. Now see what happens. Right round the angle between the base of the iris and the cornea are a number of spaces or clefts between the fibrous tissue, known as the spaces of Fontana. There is also a tiny circular canal known as the canal of Schlemm. According to some anatomists it is merely a small vein; according to others it is a lymph channel; while a third party says that it communicates directly with the Fontana spaces. I don't know which of the three is right, but it will not affect our theory. Now when pressure is put onto the fluid in the circumlental space as the lens cannot bulge backwards because there is no outlet for the vitreous, so it must bulge forwards. This is effected by some of the aqueous flowing into these spaces of Fontana, ***in fact that is the reason for their being there.*** The moment accommodation is relaxed the fluid runs back again into the chamber. In order to prove that I injected with a fine syringe a few drops of methylene blue into the aqueous chamber of an albino guinea-pig. Then I carefully watched the result through the ophthalmoscope, and when the eye accommodated, which one can readily observe, by either watching the pupil, which during accommodation should contract, or by getting someone to hold a carrot close to the guinea pig's nose, I distinctly observed the blue color stream away through the spaces in the angle, and return again immediately afterwards. Hence the increased curvature of the front surface of the lens is effected by hydraulic pressure in the way I have indicated, and not as Helmholtz affirmed. It would be well if some of my colleagues would repeat my experiments, and if they agree with mine, I think this theory will be established as a fact, and replace that of Helmholtz.

A FACTOR IN INFECTION AFTER NEEDLING.

By Dr. RALPH I. LLOYD, Brooklyn, N. Y.

NEEDLING, beside being a troublesome procedure, is now and then attended or followed by an infection that makes the operator wish for a cataract procedure which would do away with needling. While I prefer, when practicable, the removal of the lens in its capsule, this is not always wise nor does removal in capsule always do away with needling. Sooner or later, in nearly every case where proper observation can be made, the anterior vitreous layer begins to take on a gossamer like appearance if it is exposed to contact with the aqueous. This may not affect the vision so seriously as to require needling but it occurs irrespective of the type of cataract operation performed and the only reason it escapes notice in the traditional operation is because the pupil area is too small or because in the needling done, the vitreous layer was included in the proceeding. Every now and then in a successful case of intra-capsular extraction, needling is required but is of course a simple proceeding when only the vitreous contributes to the membrane formation.

After a needling recently, which was successful, the patient was allowed to go home on the third day with the eye free from every sign of trouble and reported over the telephone on the fifth day, that as soon as the patch was removed, the eye became very painful, particularly on movement. She came to the office the next day, six days after the operation, and the eye was evidently very sore but there were no signs of infection. Close inspection with strong light and the loupe, showed a fuzzy end sticking out of the wound made by the needle. This was grasped by forceps and upon pulling, more of it was brought out of the wound and upon relaxing tension, it would pull back into the eye and the internal extension of this string was seen to be continuous with the membrane left by lacerating the capsule at the original operation and which had

been needled. The whole membrane could be lifted and allowed to fall back into place. The string was pulled out as far as it would easily come and cut off but it could not be cut without leaving a bit projecting, and to avoid having a "lampwick" to carry infection into the interior of the eye, this was picked up, and a pure carbolic swab rubbed on it at the hole until it broke off, and the end in the cornea presently drew back into the eye and could be seen as projecting loose end, swaying with movements of the eye. When the needling was done, the Beebe loupe was used and the expected albuminous string which follows the end of the needle could be seen up to the cornea, but nothing was seen to project from the wound but must have done so. In the six days following the operation, the entire character of this albuminous string changed. It ceased to be a slender, transparent thread and became a string of gray, easily seen; evidently not infected, and as thick as a fine thread and as fuzzy. There was absolutely no affinity between string and cornea and it would slip in or out of the needle puncture with little resistance. A very pronounced organization of this vitreous string had taken place and could easily have been infected and then would have appeared like the well-known yellow-white line of infection from the wound of entrance down into the vitreous-aqueous junction. The Ziegler knife needle was used. A few days later, it was my privilege to assist a colleague in needling, and using the loupe, I observed the exit of the Ziegler knife needle from the wound. There was no string attached as it left the corneal surface and a carbolic acid application was made to the wound to seal it but the aqueous continued to escape in small drops for a long time and we could not seal the wound in this way.

It occurred to me that we should introduce into our routine procedures, either the sub-conjunctival introduction of the needle at the limbus to seal the wound in the cornea and the better to bite off the vitreous thread; or carefully examine the wound if we prefer the non-limbal entrance, and by some method or other, endeavor to either seal it up or effectually sterlize it. It seems reasonable to say that this fine vitreous thread can organize and act like a lampwick but carry infection instead of fluid.

TREATMENT OF TRACHOMA WITH STRONG SOLUTIONS OF SILVER NITRATE.[1]

By Dr. N. R. DONNELL, St. Louis.

I DO not wish to leave the impression that I am presenting anything new or original; indeed I did not consider that what I might say to be of sufficient interest to be made the subject of a paper until it was suggested to me.

To make one's clinical experience and the conclusions reached thereby of any value it must have sufficient background, and I believe that with twelve years of rather active practice in the lead belt, likewise the trachoma belt, of Southeast Missouri, I may modestly claim to have had some opportunity to study this disease in its many stages and phases. I doubt if many have had the opportunity, as I have had, of seeing these conditions in the home and in their native environment, where whole families are rendered helpless and dependent from trachoma. Perhaps the vision of one member of a family will be mercifully spared, that he may "pull the wild hairs" for the remainder as well as do other odd jobs about the place; such as tend a patch of corn and tobacco, or I should have said, tobacco and corn, and to take an occasional load of wood to town in exchange for a few groceries. These families live for the most part in complete isolation, in abject poverty, with no hope of relief and with that fatalistic attitude so common among these people.

Statistics reveal the rather startling fact that trachoma is the direct cause of 20% of the blindness in Missouri as well as the largest single cause of blindness, also it is the cause of as high as 50% of the blindness in several counties of the State.

[1] Read before the St. Louis Ophthalmic Society, March 30, 1923.

Trachoma is a disease of great antiquity, for we find mention made of this condition and suggestions as to its treatment in the Papyrus Ebers (written about 1650 B. C.). That there has been little essential change in its treatment for almost twenty-four centuries we readily see from the writings of Hippocrates (living 460–370 B. C.). He advocated the rubbing of the inner surface of the trachomatous eyelids with Milesian wool wound about a spindle-shaped core of hard wood. This procedure was continued until the blood ceased flowing and in its place appeared a thin watery-like fluid. This was immediately followed by the instillation of collyria which contained, as a rule, the peroxide of copper.

Silver nitrate is rather more modern, particularly in ophthalmic therapy. We are told that it was first discovered by the Probian Geber in the eighth century; that Angelus Sala in the early part of the seventeenth century was the first to recommend its medicinal use; St. Yves in the early eighteenth century was the first to use silver nitrate to any extent in ophthalmic practice. The remedy of choice in the treatment of chronic scarring of trachomatous eyelids for thousands of years (as indicated above) has been combinations of copper. It would seem from the literature that silver has been and is being used in solutions of one, or seldom to exceed two per cent., and that chiefly to check watering or suppuration of the eyes. The only reference in the literature, from a fairly searching review, to the use of silver nitrate in trachoma, with solutions stronger than two per cent., was that of Everbush in 1896. He recommended dropping a five per cent. solution of silver nitrate on the everted trachomatous eyelid, flushing immediately after with normal saline solution. Also I find a notation that Colonel Elliott, while in service in India, contracted trachoma while operating on a trachomatous patient. He was treated by the instillation of a three per cent. solution in the eyes each morning. He found that after a short time this was attended by comparatively little discomfort and that he could very soon go about seeing his patients with little or no inconvenience. This form of treatment is said to have effected a cure in five months.

My first extensive experience with trachoma was at Moorfields, London, in 1904–05 where vast numbers of emigrants

came through the clinic, thinking that a clean bill of health from that institution would assure their entrance into the United States. Hours were spent on the diagnosis, and differential diagnosis, of trachoma, but the treatment was dismissed with a few brief instructions, at that time mostly copper stick. In Vienna I found very much the same attitude toward trachoma though I saw much less of it there.

On my return home circumstances placed me in the mining industry of Southeast Missouri, a perfect hot-bed of trachoma. Trachoma-sufferers from time to time naturally gravitated to the mining centers to find employment. All those who could see well enough came there to get work, and those who could not see to work came and attached themselves to some family, where on account of a contract arrangement with the company under which I worked, they could be treated for a very nominal sum. This appalling condition was forced upon me with no alternative but to work my way out as best I could. Being early imbued with the idea that a therapeutic measure definitely indicated should, in most instances, be carried to the full therapeutic effect, I took what seemed to give the best results (namely, nitrate of silver) and soon began using it in increasing strengths. I had been taught that the cornea must be protected from the silver solution because of the bad effect, but I found that even in the presence of pannus and ulcers and with solutions of two and three per cent. most patients did well, the chief drawback being that many would not continue treatment due to the discomfort, very much of which, as I see it now, was due to my rather crude methods. I also tried rolling and sand-papering in conjunction with the solution of silver up to the point of toleration (which, by the way, differed greatly in different individuals and in different types of cases). From my experience with surgical and therapeutic measures, I began a combination of two, by massaging the everted lids with cotton wrapped tightly around a tooth pick, and dipped in a two, to five, and sometimes ten per cent. solution of nitrate of silver, all excess solution being removed from my applicator, that I could better control my application. I soon found that the use of silver in these strengths necessitated the pretty thorough cocainization of the lids before treatment, immediate neutralization by sodium chloride

solution, and the use of ice packs, which materially lessened the discomfort.

Here I would like to mention a case that came to me in 1905, a boy of about eighteen years of age, with a typical case of trachoma, with ulcers, pannus, etc., and with vision so reduced that he was unable to come to the office unattended. After treatment along these lines, of less than six months, his eyes were quiet, only a few vessels left in the cornea, lids smooth except for some scarring and vision of $\frac{1}{1}\frac{1}{1}$ in either eye. This patient since 1906 has had no treatment for, nor recurrences of, trachoma; he is now a man with a family and occupies a responsible position. I mention this case because a sufficient time has elapsed to make the results rather impressive. I could mention many others that have been equally satisfactory to me.

After this experience lasting for ten years I became associated with the late Dr. Wilson at the St. John's clinical dispensary and found that he was using, as some of you no doubt know, much the same method, only if anything was using his silver in stronger solutions. I have seen him on occasions use twenty-five per cent., and strange to say with good results, though I have never dared go quite that high.

I watched his results for about two years and must say that they were at times startlingly good, and for the most part very satisfactory.

I do not think that this method of treatment should be attempted by any but trained ophthalmologists, for at times there follows a pretty violent reaction, and it is necessary that one be able to distinguish between an exacerbation of the trachomatous process and a reaction from the treatment. One must also be able to judge when and how much to massage, and when to increase or decrease the strength of the solutions which can only be gained by experience and observation. My conclusions are that beginning with a stiff massage of the inner surface of the eyelids with a one per cent. solution repeated two or three times a week, gradually increasing in strength until the point of toleration is reached, gives the best results. Three instillations of 5% cocaine hy. solution precede all applications of silver nitrate. The acute follicular cases of course are more intractable to this form of treatment

and close attention to the strength of solution used is here necessary to prevent unfavorable irritation from too concentrated a solution. Nevertheless in the acute follicular stage of trachoma, I have found the massage with one to three per cent. solutions of silver nitrate to give quicker and more marked improvement, with less discomfort, than by any other method I have tried, which includes all the common forms of treatment for this disease. In the chronic cicatricial stage, however, the results are almost invariably good and rapidly obtained; much of the entropion and pannus with the accompanying discomfort and diminished vision can be quickly relieved in this type of cases with the exception of course of those eyes which are entirely beyond any help. The stronger solutions (2 to 10%) of silver nitrate can usually be used to best advantage on the more thickened tarsus, but sometimes even in the case of marked scarring of the conjunctiva and tarsus, marked irritation occurs with solutions of silver nitrate stronger than one per cent. Again when solutions stronger than two per cent. are used at the beginning of treatment in the marked cicatricial stage, a time is always reached sooner or later when irritation follows the application and the strength of the solution must be diminished.

Although after applications of one per cent. solution of silver nitrate to the everted eyelids, I do not immediately irrigate with normal saline before allowing the eyelids to resume their normal position, yet in the case of strengths of two per cent. and greater, thorough irrigation with normal saline immediately follows the application of the silver nitrate.

Naturally if there is any iritis as sometimes occurs with pannus and always with corneal ulceration from trachoma, atropine is used, sufficiently to obtain good dilatation.

Of course with this line of treatment, the problem that at once presents itself is that of argyrosis. This seems to be held in great horror by some. From my own experience, I can say that I have never had a marked case of argyrosis, and those where it occurred slightly, I felt, were attributable to my own lack of proper care. Attention to details, such as having fresh solutions, removing all excess from your applicator, permits very little oxidation to take place, and if it does it can immediately be detected on the white cotton of your

applicator. In that case the applicator must not be used further or staining will take place in a very few treatments.

Let me add in conclusion that I do not offer this as a cure for all cases of trachoma but in my own work the results have been so uniformly good, and so much better than anything else I have used, that I am presenting it for what it is worth.

IRIDO-CYCLITIS SYMPATHICA FOLLOWING AN IRIDECTOMY FOR GLAUCOMA SECONDARY TO SARCOMA OF THE CHOROID.[1]

By Drs. C. W. TOOKER and H. D. LAMB, St. Louis.

(With two illustrations on Text-Plate XVIII.)

SINCE Mackenzie in 1835 first accurately described sympathetic ophthalmia many cases have been recorded and valuable additions have been made to the study of its etiology, pathogenesis and treatment. Our knowledge of the disease is however incomplete. We know only that certain cases of traumatic irido-cyclitis cause at times an irido-cyclitis of a characteristic type in the fellow eye, and that the pathological condition of the injured eye has always been found to be characteristic of the disease. Inasmuch as sympathetic ophthalmia has become a rather uncommon disease it would seem proper to report in detail a case which has been studied over a period of several months. The literature regarding sympathetic ophthalmia has been so abundant that one cannot be assured that a case similar to the one herewith presented has not been previously observed. No mention by title, however, of an analogous case could be found in available references to medical literature during the last 20 years.

Mrs. B. H. S., aged 60, consulted me on Oct. 28th, 1923, because of an inflamed left eye which had begun to give her trouble on the 26th. She complained of some "scratching" and a feeling of fullness of the eyelids but there had not been any severe pain. She stated that the vision of her left eye had been failing for six months. Her health has been excellent for many years although she has always been

[1] Read before the St. Louis Ophthalmic Society, February 29, 1924.

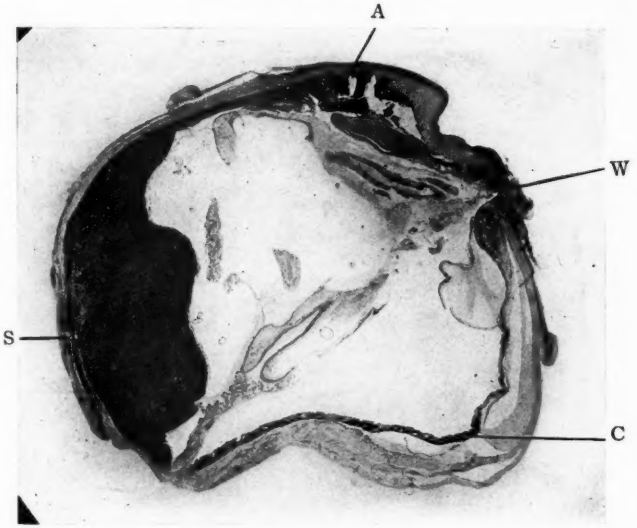
of a "nervous" temperament. She is the mother of one son who died of scarlet fever and of two healthy living daughters each of whom has borne healthy children. Neither she nor her daughters have miscarried. At the age of 50 she married her second husband. A general physical examination by an internist disclosed no abnormality. Her urine and blood pressure were normal and there were no demonstrable foci of infection in the nose and throat. All her teeth had been removed several years ago. Serological tests did not seem indicated at the first examination.

Ocular Examination Right Eye: normal, with the exception of rather sharp bending of the vessels on the nasal side of the disk. Vision equals $\frac{20}{20}$ corrected by + 1.50 D.S. to $\frac{20}{20}$; J. No. 1 at 10" w + 3.00 D.S. added.

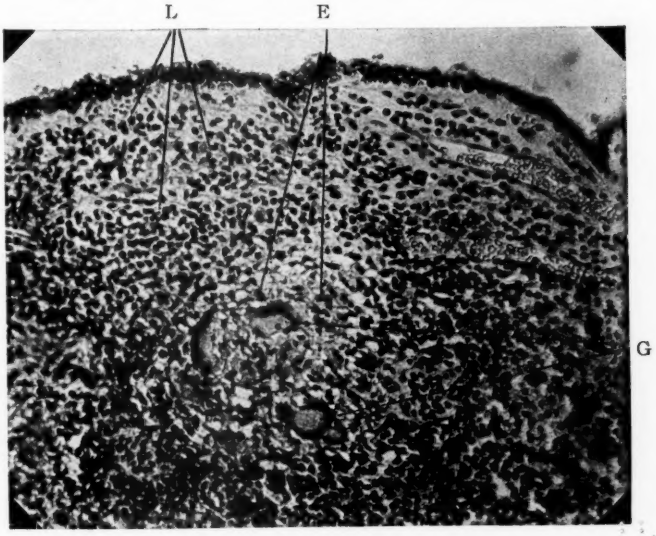
Left eye: Œdema of upper lid; chemosis of bulbar conjunctiva; marked pericorneal injection; cornea rather lusterless but not steamy; anterior chamber shallow; pupil widely dilated; no reaction of iris to light; lens completely opaque; tension 80 *mm* Hg (McLean); vision equals light perception; projection uncertain.

During the next three days eserine sulphate 2 grains to the ounce was instilled into the left eye every 2 hours after hot boric acid applications; free catharsis was maintained, and empirin was used when needed to control the pain which had developed. 1% eserine (alkaloid) in oil was used several times during daily office consultations. The pupil did not contract much and the tension remained above 55; the congestion and pain were considerably reduced. On Nov. 1, 1923, five days after the onset of glaucoma, she was sent to the hospital. An iridectomy was done at the root of the iris with a Graefe knife and a conjunctival flap made somewhat larger than is customary. The reaction was moderate and the anterior chamber was restored in 24 hours. On dressing the eye the third day following the operation the conjunctival flap was discovered rolled down. It was replaced but did not adhere to the subconjunctival tissue. Sterile 1% atropine sulphate solution was used daily following the operation and the eye kept bandaged. Ten days after the operation, the triangular area denuded of conjunctiva seemed to be covered completely by a thin membrane (not an inflammatory exudate); the ciliary injection had diminished greatly and tension measured 40*mm* Hg (McLean). The roll of conjunctiva at the upper limbus was excised and the linear stump touched with 20% solution mercurochrome 220. It was thought inadvisable at this time to further traumatize the eye by making a conjunctival apron to cover the area of operation. On the sixteenth day following the operation the patient stated that during

ILLUSTRATING DRS. C. W. TOOKER AND H. D. LAMB'S ARTICLE ON "IRIDO-CYCLITIS SYMPATHICA."



1. Vertical meridianal section through eye-ball, showing iridectomy-wound-opening (W), hypopyon (A), choroid with infiltration of sympathetic ophthalmia (C), melano-sarcoma of choroid (S).



2. Sympathetic infiltration in the choroid with small lymphocytes (L), epithelioid cells (E), giant cells (G).

the previous night she had experienced severe pain in and over the left eye. The upper lid was found to be œdematous and a hyphema was present with severe iritis. Hypopyon of moderate degree was noted on the 24th day following the operation. The patient refused consent to an enucleation. Following the iridectomy only sterile solutions had been used and the eye had been kept bandaged (wide strip adhesive plaster over cotton). Now 2% sterile atropine solution, 50% argyrol, 2% mercurochrome 220, and later 2% atropine (alkaloid) and 1% cocain (alkaloid) in oil were used. Protein injections were considered but did not seem indicated. The patient's general health continued excellent and she suffered little pain or discomfort. The hypopyon increased gradually and the irido-cyclitis was not influenced by treatment. On the 33rd day after the operation on her left eye, the refraction of her right eye had apparently changed from + 1.50 D.S. to + 2.50 D.S. giving her vision of $\frac{2}{3}\frac{0}{0}$; J. No. 1 at 10″ w + 2.25 added. On the 37th day tension in her right eye measured 30*mm* Hg (McLean); the form field was about normal, but the color fields were found to be contracted to include an area of half the normal size. The pupil was moderately well dilated and the iris reacted very slightly to light. There were no deposits on Descemet's membrane and none on the anterior capsule of the lens. Vision with correction equalled $\frac{2}{3}\frac{0}{0}$. Consent to an enucleation of her inflamed eye was obtained but the patient insisted on deferring the operation for a few days. In view of the fact that glaucoma is a bilateral affection and that one might be able to prevent an attack in her seeing eye, eserine sulphate 2 grains to the ounce was instilled in her right eye at office consultations and pilocarpin muriate 2 grains to the ounce was ordered for home use at two hour intervals. This treatment seemed the more necessary because of the expected shock to her nervous system which an enucleation might produce. During the next 3 days the pupil of her right eye gradually contracted to a width of 1½*mm* and her vision began to fail. On the 40th day after the iridectomy consultation was held with Drs. W. A. Shoemaker and W. E. Shahan without agreement as to the condition present in her right eye. There was not much ciliary injection and with a small pupil it was difficult to determine whether the minute deposits were on Descemet's membrane, although some could be seen on the anterior capsule of the lens. The iris markings were not much disturbed. Vision with correction equalled recognition of fingers at 10 feet. 2% homatropin hydrobromate was used 3 times during the night and 80 grains of sodium salicylate were given by mouth. The left eye was enucleated

on the following morning, 41 days after the iridectomy. Staphylococcus pyogenes albus grew on the surface of blood serum smeared with a particle of semi-fluid material taken from the interior of the enucleated eye under aseptic precautions. The globe was sent to Dr. Harvey Lamb for examination who reported mixed cell melanosarcoma and sympathetic ophthalmia. His complete report follows this case report.

On the day of the enucleation (the day following the tentative diagnosis of sympathetic ophthalmia), 1% atropine sulphate in solution was instilled in the right eye at 3 hour intervals and atropine has been used at varying intervals until the present writing. The pupil dilated above but synechiæ below held the iris bound to the anterior capsule of the lens. Two serologists reported 100% hemolysis (4 plus Wassermann) on the 3rd and 6th day of the disease. During the 21 days after the onset of sympathetic disease the patient received 18 daily inunctions of 1 dram of mercury and on the 8th, 15th, and 22nd day, .3 grams of salvarsan intravenously. During the same period of time 650 grains of sodium salicylate were administered at varying intervals. It was difficult to use the drug in the amounts recommended by Gifford because of toxic effects so readily produced. The patient frequently became irrational, suffered severe depression and complained bitterly of noises in the ears and mental confusion, symptoms which disappeared after two days abstinence from salicylate medication and reappeared after intensive use of the drug. She was unable to retain the salicylate given per rectum. A mild albuminuria without casts was present at times but a recent urinalysis shows normal renal function. The ciliary injection gradually subsided, the iris markings never became obscured, nor was any considerable thickening of iris tissue observed. The pupil became dilated to a width of 5*mm* but posterior synechiæ hold the iris bound below. Vision gradually improved. About 24 hours after each salvarsan injection the eye became flushed and vision was somewhat dimmed. Pigment deposit on the anterior capsule of the lens increased in amount peripherally but did not encroach upon the central pupillary area. An area of lens capsule on the temporal side was entirely free of pigment. The deposits of cells on Descemet's membrane increased in quantity during the first week of the disease but subsequently diminished in number and size. On the 27th day of the disease 1½ grains of salicylate of mercury were given intramuscularly and a similar treatment has been repeated at 4 day intervals for 5 doses. There was no reaction in the eye after these injections, but a considerable indefinite

physical and mental depression usually appeared at the end of 24 or 48 hours lasting a day or two. On the 23rd day of the disease the cornea was found to be anæsthetic and the lower third stained faintly in dots and small lines after the application of 1% aqueous solution of flourescin followed by irrigation with boric acid solution. Vision with correction (+ 2.50 D.S.) at this time equalled $\frac{20}{30}+$. For the next 10 days the eye was kept sealed for 12 hours in every 24 after the instillation of a mild antiseptic solution, and during the remaining 12 hours the following collyrium was ordered at 2 hour intervals, Atropine sulphate 8 grains, acid boric 15 grs., holocaine 1 gr. to the ounce of water. Thirty-three days after the onset of sympathetic ophthalmia V.O.D. w + 1.50 D.S. equalled $\frac{20}{20}$. Transillumination did not show the presence of a growth in the eye. The cornea did not stain with flourescin and there was no ciliary injection. The pigment deposits on the lens capsule were rather abundant but the central pupillary area was clear. Deposits on Descemet's membrane were numerous. No floating vitreous opacities have been discovered during the course of the disease but the fundus details have been obscured until recently because of deposits on Descemet's membrane and on the lens capsule and perhaps also because of a hazy vitreous. On the 40th day of the disease 1% atropine sulphate in 1–10,000 bichloride solution was ordered every 3 hours together with $\frac{1}{14}$ grain of bichloride of mercury 3 times a day internally, and this treatment has been continued to date. Dark glasses were ordered for outdoor use and a prothesis was fitted to the left socket which has remained healthy. The disk margins seemed blurred and the retinal veins rather too large. No chorioretinal spots as described by Von Graefe could be discovered.

V.O.D. w + .75 D.S. $\frac{20}{20}$.

Status Præsens Feb. 29, 1924 (80th day of disease).

V.O.D. with + .50 D.S. = + 1.00 D. cyl. ax. 90 = $\frac{20}{20}-$ J. No. 1 at 10″ w + 4.00 added.

The pupil is fairly uniformly dilated to a width of 5*mm* but posterior synechiæ hold the iris firmly bound to the lens capsule below. There is no ciliary injection, and the iris markings seem normal. Pigment deposits on the anterior capsule of the lens have decreased greatly in number and the deposit of cells on Descemet's membrane are not so numerous. Their distribution is rather peculiar; a belt of deposits can be seen crossing the cornea below its center but the lower limbus is clear. The vitreous is not clouded and the outlines of the disk are distinct. The retinal arteries and veins seem to be of normal size and relation. No chorioi-

dal spots can be discovered in the fundus. The eye is not sensitive to pressure and the patient does not complain of any pain or discomfort in the use of her eye. She has not been permitted to use the eye for reading or sewing.

COMMENT.

Some suspicion of the presence of a tumor in the glaucomatous eye might have been suggested by the history of a unilateral disorder, by the absence of much pain, and by the uncertainty of projection, but an enucleation did not seem warranted until irido-cyclitis had developed. The failure of the flap of conjunctiva to adhere to the globe may have been due to its size, to the diseased condition of the eye or to both. There was no primary infection at the time of operation. The staphylococcus infection 15 days after the iridectomy seems to be an example of late infection through a poorly covered operative area notwithstanding the careful attention to details of dressings and solutions used in the eye. It is of course possible that infection resulted when the roll of conjunctiva was cut off. The fine shred of capsule shown in the microscopic sections may have retarded the healing of the wound. This shred may however be an artifact in mounting and sectioning the eye. A belief in the theory of the endogenous origin of sympathetic ophthalmia offered by Meller, Boehm (1) and others would lessen the burden of responsibility in a case of this sort, but the facts seem to point conclusively to the occurrence of a late secondary infection at the site of operation. Collins (2) states that the staphylococcus is the usual bacterium found in the offending eye and that it seems to have the power of circulating in the blood without producing much general change. The theory of a bacteræmia of low general virulence or of a uveal sensitization must be purely speculative until an examination of the sympathizing eye can be made. About one year of life may be anticipated by those in whom mixed cell melanosarcoma has been discovered. The influence of sarcoma itself in the pathogenesis of sympathetic ophthalmia has been the subject of considerable controversy. Schirmer, Milles and Deutschmann (3) reported many years ago cases of intra-ocular tumor causing sympathetic ophthalmia. Meller (4) and A. Fuchs (5) more

recently have discussed similar cases. Necrosis in the sarcoma may be the necessary factor according to Schirmer. The association of lues and sympathetic ophthalmia has been studied by K. Ichikawa (6). In an examination of 7 eyes removed from leutic patients because of sympathetic ophthalmia he could find no spirochetes. The presence of syphilis, sarcoma, secondary infection and sympathetic disease in this case rather confuses etiological conclusions.

The clinical diagnosis of sympathetic ophthalmia presented some difficulties. Three days before the enucleation of the inflamed eye one would not have been surprised at the development of glaucoma in the seeing fellow eye. Certainly no definite signs of sympathetic irido-cyclitis were present. The possibility of such a condition had been considered often in consultation. In the evening before enucleation one of the consultants thought that there might be present a mild anterior uveitis of independent origin, or that the use of eserine for several days had produced a severe reaction in the iris and ciliary body. No deposits on Descemet's membrane could be defined with certainty although there was a marked clouding of the anterior capsule of the lens with deposits of pigment and cells. Fuchs (7) is emphatic in his statement that deposits on Descemet's membrane are never wanting in the early development of sympathetic disease and many observers agree with him in that opinion. Ellett (8), however, reported a case recently in which deposits on Descemet's membrane did not occur early. The examination of the enucleated eye did not entirely substantiate the clinical diagnosis. Many writers state that sympathetic ophthalmia is seldom found in elderly people and it is in this class especially that glaucoma develops. Although dimness of vision is regarded as the earliest sign of sympathetic disease the partial loss of vision in this case could be explained by the apparent development of glaucoma; dilated pupil (not produced by medicine), contracted color fields, and suspiciously sharp bending of the retinal vessels at the nasal margin of the disk. Even though the tension seemed to be within normal limits additional emphasis was of course given these findings because of the history of glaucoma in the eye to be enucleated. The decision as to early treatment was naturally difficult. If

sympathetic ophthalmia were beginning, atropine ought to be exhibited freely but should the eye be glaucomatous, the use of atropine would result in disaster. Homatropin was therefore used at first with the idea of controlling its action with eserine later if necessary. The examination of the enucleated eye, the report of which came a week later, established the diagnosis, although according to Fuchs the diagnosis of sympathetic ophthalmia should be made only if the irido-cyclitis is purely a local one and not dependent on constitutional causes. Syphilis in this case probably lowered the patient's resistance to trauma and to infection, and may have made the uveal tract of the seeing eye "sensitive."

The treatment was of such a mixed character, intentionally so because of the anxiety to save some sight, that opinions as to the efficacy of any one remedy cannot be of much value. Mercury has been recommended by Mackenzie and many others, and more recently Fleisher (9), Browning (10), Siegrist (11) and others have reported favorable results from the use of salvarsan. The favorable outcome in this case may perhaps be attributed to these two remedies. The total quantity of salicylates used was much less than that recommended by Gifford. Good results have also followed the use of tuberculin according to Bernheimer (12), Stoewer (13), Norman (14) and others. Derby (15) considers that the favorable result in two of his cases is due to the use of a serum prepared from the blood of a patient suffering with sympathetic ophthalmia. In these cases, however, other remedies also were used in large amounts. It would seem that in many cases no treatment is effective and that recovery sometimes follows the use of a combination of remedies.

It was thought in former years that as a rule these sympathizing eyes ultimately became blind, but Gifford (16) states that if seen within the first week and properly treated 75% of the cases retain useful sight. The condition of the eye in this patient is at present, therefore, satisfactory. If the eye can be obtained post-mortem a comparison of its pathological condition with that given in the following report would prove interesting.

PATHOLOGICAL REPORT, BY DR. H. D. LAMB.

Macroscopically the eyeball of Dr. Tooker's case examined for the first time after being in 10% formalin for two days showed no shrinking, cornea was densely opaque with a greyish-white infiltrate and through an open wound at the superior limbus there protruded a small amount of dirty gray membrane.

After sectioning the eyeball just to one side of the optic nerve posteriorly and the open wound anteriorly with a sharp razor examination of the cut surface revealed the following:

Anterior chamber was very shallow; the lens represented as a small thin flat disk was located forward and somewhat downward away from the limbic wound; the anterior chamber was quite filled with coagula, whereas the vitreous chamber was fairly free from them; the retina was completely detached and posteriorly near the optic nerve there was a dirty grayish-white mass of quite firm consistency.

From the smaller segment of the eyeball the posterior mass with the choroid and ciliary body on one side was detached and excised. This piece was separated into approximately halves, dehydrated, imbedded in paraffin, sectioned and stained with hematoxylin and eosin. The time to accomplish this consumed but four days and thus a fairly prompt report of sympathetic uveitis and melanosarcoma of the choroid could be rendered.

The larger segment of the eyeball at the same time was being more slowly dehydrated, imbedded in celloidin, sectioned and stained with hematoxylin and eosin. These sections are vertical longitudinal ones of the eyeball as it lay in the orbit.

Macroscopically the prepared and stained celloidin section, through the operative wound, shows in startling relief a dense mass posteriorly and inferiorly in the eye colored dark blue by hematoxylin—the nuclear stain. The choroid on both sides of the section stands out as a thin dark blue line of nuclear staining. Anteriorly the ciliary body on both sides and the iris on the side opposite the iridectomy wound can be easily located by reason of the greatly increased amount of dark

nuclear staining in their structure. The sclera posteriorly and the cornea anteriorly has been drawn inward a little, a sign of shrinking from the dehydration in the alcohols. The margins of the iridectomy wound and in the wound opening itself superficially there is seen some dark staining signifying again a cellular infiltration. The same appearance in the form of small dark blue streaks is found in the sclera anteriorly and posteriorly on each side of the section indicating a cellular infiltration around the anterior and posterior ciliary vessels. Due to the lens remains being pushed forward particularly on the side of the operative wound, this open corneal wound is seen communicating directly with the vitreous chamber. Some dark-staining evidently of nuclear material can be seen in the lower part of the anterior chamber or on that side opposite to the limbic wound; this must signify the presence of a hypopyon. Posteriorly the unthickened completely detached retina can be seen in rather broken pieces appearing in a greatly wrinkled and folded condition. A prolapse of several retinal folds into the limbic wound can be made out as well as the remaining attachment of the retina to the ora serrata on each side and the optic nerve behind. Prepared sections from deeper in the block, in other words going farther laterally in the eye the dense dark blue stained mass posteriorly and below increases in size. A second lobe of similar appearance is seen, in the mounted sections, just anteriorly to the first mass; the two lobes are completely separated from each other except for a narrow isthmus or neck adjoining the outer wall of the eyeball. In some sections the area of the two lobes, together, occupies about two-fifths of the area of the eyeball.

Miscroscopic Findings.

The cornea is found without change except in that portion which adjoins the operative wound. Here it increases gradually in thickness until at the wound margin it is about three times thicker than the normal depth of the cornea at this locality. This thickening is mostly due to œdema between the corneal lamellæ, although there is also present a moderate amount of infiltration between these lamellæ with small

lymphocytes, eosinophiles and plasma cells which are diffusely scattered through this portion of the cornea. Superficially, near the corneal margin, the anterior epithelium is seen to have a large epithelial downgrowth. Adjoining this point and lying between the conjunctival epithelium and the superficial corneal lamellæ there is a very dense focus of mostly small lymphocytes but with some eosinophiles, plasma cells and many capillaries; this focus extends to the wound margin. An exudate composed mostly of eosinophiles and plasma cells but also some red blood cells and lymphocytes is found lining the wound margin on the corneal side. This exudate is covered by epithelium, which is a reflection of the conjunctival epithelium, downward over the wound margin to a considerable depth although but a few cells thick for about the lower half of its extent. Along this margin of the wound and imbedded in this exudate there is seen a much wrinkled loop of lens capsule, almost white in color.

The opposite or scleral side of the wound shows the sclera but little thickened but superficially adjoining the wound margin there is a large mass of cellular exudate and infiltration composed of eosinophiles, plasma cells, small lymphocytes and red corpuscles.

In the wound opening the prolapsed retina shows marked degeneration and slight infiltration with lymphocytes, eosinophiles and plasma cells.

The anterior chamber is filled anteriorly with coagulated serum, posteriorly with a cellular exudate. The latter on the side of the iridectomy wound is in some sections a circumscribed elongated and dense mass of eosinophiles, plasma cells and lymphocytes in about equal numbers. This is located just anterior and in contiguity with the lens remains and extends over as far as the pupillary margin of the opposite side of the iris and even beyond posterior to it. Anterior to and resting on the iris on the side opposite to the iridectomy wound, in other words in the lower part of the anterior chamber, there is a large hypopyon but composed entirely of eosinophiles, both polymorphonuclear and mononuclear (plasma cells). The iris angle is closed on this side by a rather wide extent of the base of the iris having been pushed forward and outward against the sclera and cornea. The meshes of the

ligamentum pectinatum are to a moderate degree covered with eosinophiles and plasma cells; Schlemm's canal and the radicles of the anterior ciliary veins are filled full with red corpuscles.

The iris is but a little thickened; it is diffusely and densely infiltrated throughout its extent with small lymphocytes mostly, but also with a considerable amount of eosinophiles and plasma cells. Mostly in the posterior part of the iris, there are seen a number of large and small lighter staining islands, containing epithelioid cells or some epithelioid and a giant cell or two. The epithelioid cell is here a large cell with a large round or oval lightly staining nucleus in a large amount of lightly staining cytoplasm. The entire thickness of that part of the iris which lies against the sclera is moderately infiltrated with eosinophiles and plasma cells only. The posterior pigmented epithelium shows loss of continuity in many places; quite frequently here and there these pigmented cells have wandered forward into the iris-stroma and appear in small groups or as single isolated cells; in either case each cell is always round and densely pigmented. The blood vessels of the iris, which are small arteries for the most part, show in many places a characteristic change. The vessel-wall has become thickened to a moderate degree, followed by a hyaline degeneration of this wall; finally an infiltration with lymphocytes and a gradual obliteration of all vessel-remains.

The ciliary body is generally thickened, in some sections to two or three times the normal thickness. It is not infiltrated as much as is the iris; the degree of infiltration observed in different sections varies greatly; in fact this amount varies considerably in the ciliary body as seen on the two sides of the same section. The infiltration is generally greater on the side of the tumor-mass. This infiltration is generally diffuse, although there are seen round or elongated foci of infiltration in some of the sections. The composition of this infiltrate is mostly small lymphocytes, although there are seen in addition many eosinophiles and plasma cells diffusely scattered and in the anterior portion of the ciliary body the latter are the preponderant cellular forms. Generally on the inner side there occur here and there islands of epithelioid cells with an occasional giant cell. The ciliary processes show little infiltration

but do have considerable fibrosis and hyaline degeneration at their ends in some places.

The choroid is generally thickened to several times its normal depth. In those parts not involved by the neoplasm it shows the most marked cellular infiltration of all the uveal structures. Even in the anterior portion of the choroid, where the involvement is much less intense than posteriorly, there are not many spots, where there is no cellular infiltration; here the infiltration occurs more generally in foci with less infiltrated areas between. As we proceed posteriorly the infiltration very soon becomes very intense throughout the thickness of the choroid as far inward as the choriocapillaris; in a very few places even this is involved and the lamina vitrea is the internal limiting structure. The cells forming the infiltrate in the choroid are small lymphocytes in much the greatest proportion, but also there is present a moderate amount of eosinophiles and plasma cells diffusely scattered throughout the masses. A few light-staining areas of greater or less extent are seen at irregular intervals composed of epithelioid and an occasional giant cell. The lamina vitrea and pigmented retinal epithelium are but little changed. The suprachoroidea shows a rather general infiltration with the prevailing form of cells. On the side of the neoplasm but in that part unaffected by the tumor the choroid shows the greatest thickening of that membrane that appears anywhere in the sections. The infiltration however is not marked in this locality except near the neoplasm where there are seen many lymphocytes and a great proliferation of chromatophores.

The bilobed tumor shows in its posterior lobe mostly spindle cells with a comparatively small amount of pigment; the anterior lobe however is composed mostly of small round cells and contains a great deal of pigment. A mixed celled melanosarcoma of the choroid would probably be the correct designation of this growth.

The anterior and posterior emissary blood-vessels and nerves in the sclera show in most places a marked enveloping infiltrate of lymphocytes with a few eosinophiles and plasma cells; this prevails also in the sclera covering the sarcoma.

The completely detached retina shows throughout a marked degeneration but practically no infiltration.

Comments on the Pathological Findings.

In the pathological findings of this case there are two features of unusual interest. The first is the absence of practically all plastic exudate on the posterior surface of the iris and on the inner surface of the ciliary body, the presence of which is so characteristic of sympathetic uveitis. In this connection Fuchs states (*Archiv für Ophthalmol.*, 1905, Vol. 61, p. 365) that this plastic exudate is not always present in sympathetic disease. He is of the opinion that the plastic exudate is to be considered as a part of the traumatic endophthalmitis and is distinct pathologically from the sympathetic uveitis. The former is the well-understood septic intraocular condition which can follow any perforating wound; the sympathetic uveitis is the characteristic infiltration in the iris, ciliary body, and choroid due to some unknown toxin. It is probable that because of this absence of plastic exudate, cellular precipitates did not occur on Descemet's membrane in this case.

The second feature of unusual interest is the presence of the mononuclear and polymorphonuclear eosinophile cells composing the hypopyon and with the small lymphocytes forming the infiltration in the margins of the limbic operative wound; they are the cells which are scattered in moderate numbers throughout the sympathetic infiltration of the uvea; the commonly present neutrophilic polymorphonuclear leucocyte is entirely absent, as is likewise the ordinary plasma cell. Andreas Rados in an article on "The Presence of Eosinophile Cells in the Eye" (*Archiv für Ophthalmol.*, 1920, Vol. 103, p. 331) states that a number of writers have reported the presence of mononuclear and polymorphonuclear eosinophile cells in sympathetic ophthalmia. A. Fuchs (*Archiv für Ophthalmol.*, 1920, Vol. 103, p. 228) thinks that the mononuclear cell with eosinophile granules is a degenerated derivative of a plasma cell.

I wish to add that the sections in this case were prepared at the Eye Research Bureau maintained by the Missouri Association for the Blind and the St. Louis University Medical School.

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REPORT OF A CASE OF CHLOROMA OF THE ORBIT.[1]

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(*With four illustrations on Text-Plate XIX.*)

CHLOROMA or leucosarcomatosis is a definite clinical and pathological entity, presenting the blood picture of an acute myelogenous (or rarely lymphatic) leukæmia together with the formation of infiltrating tumors, usually but not necessarily of a greenish color, composed of pathological blood elements, in various parts of the body, but with a predilection for the bones of the skull, particularly of the orbit.

Although as far back as 1821 Allen Burns reported a greenish tumor of the orbit, and isolated cases of a like nature are listed in 1831, '36, and '54, variously described as green cancer, fibro-cellular and fibro-plastic tumors, even Virchow, in 1866, classifying them as sarcoma, it was not until 1878 that Huber made the first blood examination and discovered a then unknown picture which he was sure had some connection with the tumor, recognizing as he did, the blood elements in the growth.

Waldstein, in 1885, first recognized as an entity leukæmia with bone tumor, which he called chlorolymphom, and this was verified by the exhaustive studies of Recklinghausen and by Dock in 1893; the latter also proved conclusively that the tumor was not sarcoma. Further studies by Sternberg in

[1] Read before the Section on Ophthalmology of the New York Academy of Medicine, Feb. 19, 1923.

ILLUSTRATING DR. CYRIL BARNERT'S ARTICLE ON "CHLOROMA OF THE ORBIT."

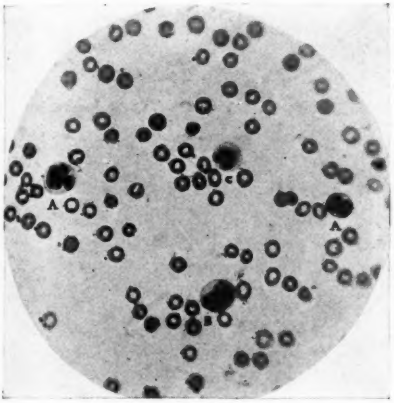

FIG. 1.—Blood smear, showing two so-called "chloroma cells" (A), myeloblast (B), and polymorphoneuclear neutrophile (C).

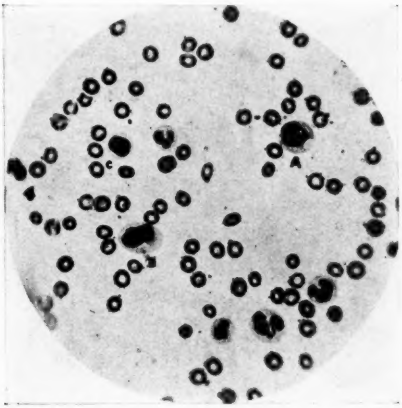

FIG. 2.—Blood smear showing myeloblast (A) neutrophylic myelocyte (B), normal lymphocyte (C) for comparison, and four polymorphoneuclear neutrophiles.

FIG. 3.—Section of orbit tumor showing marked leukæmic infiltration.

FIG. 4.

1904, who termed the condition chlor-myelosarcomatosis, and of Paltauff in 1896 under the term lympho-sarcomatosis have confirmed the fact. Nevertheless the first authentic case diagnosed in life was that of Dunlop in 1902 in an emaciated man presenting hemorrhages in the skin, deafness, bilateral exophthalmos, enlarged glands and a blood picture of Hg 32, R.B.C. 1,800,000, W.B.C. 24,500, Lymph 75%, Polys 17, unclassified 5 and a few nucleated reds.

Incidence: Although Dock and Warthin, in 1904 refer to but 22 indubitable cases, Lehndorf, in 1910 reports the study of 90 cases in the literature, of which 20 are excluded because of lack of authenticity. In Lehndorf's résumé of 70 cases, 34 were under 15 years of age, 34 between 15 and 30 years; the youngest was 10 months, the oldest 68 years; 50 were males. In Dock's series of 22 cases, 19 were males. The average age was 18.8 years, 15 cases less than 25 years, 12 less than 16 years and one was 10 months old.

Duration: The disease is essentially fatal. The average duration is 3 to 4 months (Lehndorf), 5.5 months (Dock). Some cases were fatal in a few weeks and one record of 1½ years duration has been found.

Etiology: No evidence of an hereditary influence is substantiated. Trauma, as in our case, followed in a day or so by the appearance of tumor and a definite leukæmia has been noted but is considered as a coincidence in calling attention to a pre-existing condition. As there is recognized, however, an acute traumatic leukæmia, such a connection may exist also in chloroma. The process was regarded as an acute infection by MacCullum.

Symptomatology: Chloroma manifests itself usually with the appearance of exophthalmos, rapidly increasing, accompanied by diminution of visual acuity, occasionally by pain in the ear and loss of hearing. The progress is rapid, new tumors appear, especially in the bones of the skull. The younger the patient the more rapid the course. The leukæmia is now marked and purpuric manifestations appear in the skin and the mucous membrane of the mouth and throat, and there may be bleeding from the intestines. There is rapidly progressive secondary anæmia, with attendant weakness, pallor and evidence of exhaustion. The liver is usually enlarged, as are also the

lymph glands. There is tenderness on pressure over the long bones, and tumors may be found in any or all of the viscera. The skin is commonly the seat of circumscribed or diffuse tumors. The spongy bones are a favorite site for tumor formation, but surprisingly there is no record of spontaneous fracture. The cranial nerves are frequently involved. Icterus is rare. In Dock and Warthim's series of 22 cases, weakness in 9, pallor in 11, exophthalmos in 11, deafness in 10, swelling in the temporal region 9, in the glands 8, hemorrhage in skin or mucous membrane 8, blindness in 4. Pain is often pronounced. The temperature is often septic in type especially toward the end of the disease, but may be normal. The sensorium is usually clear.

Associated Conditions: Tuberculous glands 3 cases. B. Tuberculosis in tonsillar crypts 1, (Schmidt, Rosenblath, and Koener). A case is reported of suppurative otitis media with mastoid symptoms which on operation showed a green tumor of the bone.

Hematology: The blood picture is usually that of acute myeloblastic leukæmia. Cases formally reported as lymphatic may be in error due to the gross similarity of lymphocytes and myelocytes. MacCallum (Path. pp. 852) states, "These cases closely resemble acute lymphoid leukæmia, the difference being that the non-granular mononuclear cells are not lymphocytes, but myeloblasts as shown by their possession of proteolytic fermentation in an alkaline medium and by their positive oxidase reaction." This view is held also by Nathan Rosenthal. MacCallum nevertheless classifies chloroma as of two types, one with a predominance of lymphoid cells and one with a predominance of myeloid cells, and Naegeli also claims there is an identical infiltration of the tissues in lymphatic leukæmia. The myelocytes may reach 32% (Pope and Reynolds). A type cell named by Libman the chloroma cell is probably a transitional myeloblast. Other blood changes regularly observed are a rapid diminution of the blood platelets, thus accounting for the purpura, and of the hemoglobin. The white blood cells vary in number from 4,500 to 124,000 or more. Cases are reported in which the tumor was wel developed before any changes were noted in the blood (Sternberg, Paltauf and Buschke).

General Pathology: Chloroma is essentially a malignant disease of the bone marrow, the type cell being the myeloblast and its derivatives. Tumor-like growths appear within the bone marrow, extending through the cortex to spread over the periosteum. Some spring apparently from the periosteum itself. Some of these tumor cells escaping into the blood, not only carry the disease to other parts of the body but actually metastasise in the blood stream itself, a unique process, producing the leukæmia. When rapidly growing the recognition of the tumor by morphologic characteristics alone may be difficult or impossible owing to the diversity of the cells found. The involvement of the other bones is so rapid that no data are available as to the original site in any given case.

There is early involvement of the spleen, liver, lymph nodes, kidney, adrenals, cranial bones, vertebral column, sternum and other bones. The bone marrow may be chocolate colored or green (Meixner). As to distribution of the chloroma masses, of 28 authentic cases studied, tumor masses were found in some part of the head in all but 2 cases; in the dura 2, adherent to lids but not to periosteum 2, naso-pharynx 1, orbit 13 (periosteum), sinuses 9, temporal bone especially auditory region 7, temporal fossa 9, sphenoid 3, ethmoid 2, choroid plexus 2, vertebræ 9 (usually in periosteum of bodies), sternum 7, ribs 8, bone marrow 8, liver 9, kidneys 13, spleen 4, stomach 1, intestines 4, appendix 2, adrenals 1, lymph glands 12. Also occasionally in nose, maxillary antrum, pterygoid fossa, soft palate, gums, mastoid cells, epiglottis, larynx, aretnoid cartilage, thyroid, thymus, bladder, prostate, urethra, ovaries, mammæ, pancreas, diaphragm, pelvic connective tissue and periosteum, insertions of many muscles, sacrum, coccyx, iliacs, diploe of skull, brachial plexus, adventia of all coats of arteries and veins, subcutaneous tissues and skin. The thoracic viscera are rarely involved, but occasionally masses are found in lungs, pleura, pericardium, endocardium, myocardium. A. Plaut of Hamburg, described to the author an autopsy in which he called attention to the green hue of the fat of the abdominal wall, and a chloromatous mass was subsequently found in the skull.

The color: The typical chloroma color is a light olive green

rapidly fading to light yellow green and disappearing after several minutes exposure to light. The color may be restored by peroxide of hydrogen or ammonia. Absence of color does not affect the diagnosis, the tint is often absent in some of the masses or even in parts of the same mass. Birk and Lehar report two typical cases without color. Beitzke noted an absence of color in the tumor in an exploratory incision where the section showed it present. In our case 2 sections of the orbital mass were colorless, but at autopsy a deeper portion of this mass was green. The color has been variously ascribed to iron, to chlorophyl (the green protoplasm of plants), and to fat globules in the type cells. Whatever the chemical cause, the color is undoubtedly due to the preponderance of undifferentiated myeloblasts. Mallory states that if the blood in myelogenous leukæmia is rapidly centrifuged with as little exposure to air as possible, the layer of leucocytes presents a greenish yellow color, the depth of color being dependent upon the proportion of myeloblasts.

Case Report

C. H. I., Jr., boy, 2 years of age, family history and previous history devoid of interest.

12/16/22. *Present Illness:* On November 12, 1922, the child, then apparently in perfect health, came home crying, saying that he had been hit in the face by another child. The same evening the boy collided violently with his sister, bumping his head. No injury was noted at the time, but the following morning swelling of the upper left eyelid was noted, which persisted and increased gradually, accompanied by a slight bulging of the eyeball. There was apparently no interference with vision. The protrusion of the globe increased for ten days and then remained stationary, according to the parents. On November 25, 1922, the child was examined by Dr. H. S. Willard of Paterson, N. J., and X-rayed by Dr. H. N. Goldberg of the same place, the latter reporting the roof of the left orbit apparently elevated and a sight haziness in the posterior portion thereof. The symptoms persisting, the boy was referred to Dr. Harold Hays and in turn to the author, on November 29, 1922, at which time the following condition was noted.

Child well nourished, but pale and rather listless. Considerable puffiness of the left eyelids, especially the upper, marked proptosis of the left globe, measuring *22mm* at 87 as compared with 16 in the right eye, with limitation of

motion up and out. The edge of a doughy mass was felt above the globe extending back into the orbit. The eye was otherwise normal except for engorgement of the retinal veins, and the vision was at least $\frac{1}{1}\frac{0}{0}$, with Snellen pictures. The right eye was normal throughout, the vision being $\frac{1}{1}\frac{0}{0}$. An X-ray examination of the head made the same day was reported as showing a fissure fracture of the frontal bone, involving the orbital plate, the orbital margin, and zig-zagging upward and toward the median line for several inches.

Upon this history, examination and report, a provisional diagnosis of hæmatoma of the orbit was made. Further examinations showed a gradual increase in the proptosis and limitation of motion until the exophthalmos measured *28mm* one week later, after which further measurements were impossible due to the difficulty in locating the outer orbital margin. At the same time a diffuse area of deep ill-defined œdema or mass was noted over the right malar region. On December 9th, the left optic nerve head showed beginning œdema, which gradually increased and was accompanied by one or two retinal hemorrhages. Ecchymosis of the right lower lid first appeared at this time. On December 13th chemosis at the inner canthus of the left eye appeared and persisted. On December 15th, ecchymotic spots appeared in the right lower lid and palpation disclosed the edge of a mass, similar to that felt in the left orbit, just above the right infra-orbital ridge. Also a puffy mass over the left malar was observed. The question of new growth having been raised, the child was admitted to the service of Dr. Chas. H. May at Mount Sinai Hospital for exploration of the orbit.

12/16/22. *Examination:* Left eye marked proptosis, the eye pushed forward, down and out, with limitation of motion upward and in. There is corresponding bulging of the upper lid, with some œdema of both lids. The lower lid shows an area of ecchymosis. Palpation reveals a definite mass, fairly solid, occupying the space between the proptosed globe and the supra-orbital ridge, extending backward into the orbit. The cornea is clear, the iris under mydriasis, the media clear, the retinal veins markedly engorged, the nerve head congested and elevated about two diopters, its outlines blurred. There is a small retinal hemorrhage above and below the disk. The ocular conjunctiva at the inner canthus is chemotic. Right eye—Slight puffiness of lids, with ecchymotic spot in lower lid. Above left infra-orbital ridge is felt the edge of a mass similar to that in the left orbit. Cornea clear, iris dilated with atropine, media and fundi nomal.

The veins adjacent to the left orbit are dilated, and a rather hard ill-defined swelling or mass is seen and felt over each malar region.

12/16/22. *Operation:* Exploratory section of the left orbit.

General anæsthesia.

Incision, *2cm* slightly curved, parallel to and *1cm* from outer edge of supra orbital ridge, through skin subcutaneous tissue and tarso-orbital fascia, disclosed firm mass apparently new growth white in color, nodular. A small piece was resected for microscopical examination, and the wound closed with three silk sutures.

12/16/22. General Physical Examination. Dr. Murray Bass.

No abnormalities nor masses noted except those in the head above described.

12/19/22. Right eye appears slightly proptosed. Indefinite sensation as of mass over right globe. Fundus and media right eye negative. Left eye-wound clean, some subconjunctival and subcutaneous hemorrhage.

12/19/22. X-ray treatment begun. 10 min 9 in dist vacu. w 9 in gap 7 ma *6mm* filter. S. STERN.

12/20/22.

Hgb.	50%	Myelocytes	9.5%
R.B.C.	2,904,000	Myeloblasts	16.0%
W.B.C.	124,000	Myelogones	5.0%
Platelets	15,000	Lymphocytes	25.5%
Polys, N.	30.0%	Monocytes	13.5%
" E.	.5%	Normoblasts	4 per 200 W.B.C.

Acute myeloblastic leukæmia. Dr. Nathan Rosenthal.

12/20/22. Report of examination of section—Dr. Fred S. Mandlebaum.

Fibrin elements only, no evidence of malignancy.

Operation: Exploratory section left orbit.

General anæsthesia. Previous orbital wound re-opened and enlarged nasally. Tarso-orbital fascia incised, exposing hard mass in normal location of the lacrimal gland. This was removed for examination, disclosing a white homogeneous mass infiltrating the orbit. Piece removed for examination. Orbital periosteum covering a hard mass incised, mass removed. Wound closed with three silk sutures. Bleeding not unusual, easily controlled by simple pressure.

12/22/22. Axillary glands moderately increased in size, especially on right side. Hemorrhagic spots on arms and legs.

12/22/22. Citrate transfusion—200*cc.* No reaction.

Schick test—positive; 75 units diphtheria injected into abdominal wall.

12/24/22. Right eye seems less proptosed and orbital mass less marked. Puffiness over zygomata less marked. Fundus normal. Left eye, small ulcer lower ⅓ of cornea. Proptosis less marked.

12/24/22. Report of sections—Infiltrating leukæmic tumor; leucosarcomatosis. Dr. Mandlebaum.

12/25/22

Hgb.	65%	Myeloblasts	32%
W.B.C.	113,000	Lymphocytes	30%
Polys, N.	29%	Monocytes	3%
" E.	1%	Normoblasts	1%
Myelocytes	9.5%	Myelogones	1%

12/26/22. X-ray treatment orbit.

12/30/22. Small area of ecchymosis in right upper lid and in right frontal region. No mass felt in left orbit. Exophthalmos receding O.S.

1/2/23. X-ray treatment.

1/5/23. Temperature 105. General examination negative.

1/6/23. Purpuric spot in right external auditory canal. General examination negative. Temperature 104.

1/8/23. Hgb. 40% 10,000 platelets
R.B.C. 2,240,000
W.B.C. 72,000 Differential as above

Blood culture negative. Temperature ranging from normal in A.M. to 103.5 in P.M. General examination negative—Dr. Schwartz.

1/9/23. No signs in chest to account for temperature. Suggest blood culture—Dr. Schwartz.

1/10/23. Blood culture—negative.

1/12/23. Child is stuporous, roused with difficulty, appears very weak. Pallor more marked. Several fresh areas of purpuric spots above left orbit and forehead. Anterior posterior cervical glands enlarged. Lips, buccal and pharyngeal mucous membranes show dry ecchymotic spots and clots.

1/13/23. Stuporous. Hyperpyrexia. Pulse weak. Diffuse purpura left orbital region.

1/16/23. Extreme pallor. Diffuse ecchymosis forehead and cheeks. Pulse barely perceptible. Exophthalmos entirely gone.

1/16/23. 3 P.M. Ceased to breathe.

REPORT OF AUTOPSY.

Hospital No. 225666. Name: C. I., Jr.
Admitted: Dec. 16, 1922. Age: 2½ years; Ward X
Died: Jan. 16, 1923. Time of death: 3 P.M.
Nature of Examination: Abdominal and cranial incision
Performed by: Dr. Friedman, Jan. 16, 1923, 7:45 P.M.

General: Body is that of a young male child about 2 years old. Rigor mortis incomplete. No petechiæ, no jaundice. There is diffuse purpuric eruption over the forehead especially over the left orbit. There are no other areas of hemorrhage in the skin. The lips are covered by numerous areas of dry blood. The anterior and posterior cervical lymph nodes are slightly enlarged. The other superficial lymph nodes are not enlarged. There is a moderate amount of swelling around the left orbit. Along the superorbital line on the left side there is a recent scar measuring about *2cm* in length. There is no fracture of the orbital bone. The long bones of the body, the cranial bone and the ribs show no tumor formation. The left orbit shows a small mass behind the globe, *1cm* × *5cm*, the posterior surface of which is green, the rest being white. The right orbit is negative.

Chest: The thymus is replaced by a good deal of fat. There is no evidence of tumor formation in it. Trachea contains a moderate amount of pinkish frothy fluid. Mucosa is reddened and œdematous.

Œsophagus: Is negative. There are no masses found on the posterior surface of the sternum. Both pleural sacs are free of fluid and adhesions. Tracheo-bronchial lymph nodes are not enlarged. On section, some of them have a bright greenish homogeneous appearance.

Lungs: Together weigh *750gms.* Beneath the pleura of both lungs near the hilus there are several areas of greenish streaking which appear to be in the lymphatics. Lungs show moderate congestion and œdema.

Heart: Pericardial cavity contains a normal amount of pericardial fluid. Surfaces are smooth and glistening. At the apex of the right auricle there appears, in the pericardial surface, a small greenish area measuring about *1mm* in diameter. Heart valves are negative. Heart muscle is light brown in color. Beneath the endocardium is seen a great deal of fatty streaking. The coronary arteries and aorta are negative.

Abdomen: Panniculus adiposus is abundant. Peritonaeal cavity contains about *500cc* of brownish, non-odorous fluid. The serosa of the large and small intestines are spotted by numerous pinpoint reddish areas. The retro-

peritoneal and mesenteric lymph nodes are moderately enlarged. On cut section they have a light greenish homogeneous appearance.

Liver: Capsule is smooth. On cut section, the surface has a light greenish appearance. This is especially true about the biliary ducts. Liver parenchyma shows considerable fatty infiltration. The central veins are barely visible. The gall bladder contains a large amount of light green fluid. The bile ducts and blood vessels are negative.

Spleen: Is enlarged. Capsule is smooth and covered in several places by a shaggy fibrino-purulent exudate. On cut section, the spleen has a light reddish color streaked by numerous light yellowish areas. Malpighian corpuscles are not visible. Blood vessels are negative.

Adrenals: Both adrenals appear to be negative, but on cut section the entire cortex and medulla have a light greenish color. About the left adrenal is a mass of light yellowish fleshy material.

Kidneys: Capsule strips with ease revealing a light green surface upon which is found numerous large and small areas of hemorrhage. On cut section, there is normal proportion of cortex to medulla. In some places the surface has a deeper greenish color than the rest of the tissue. Both pelves contain a large amount of blood, and there is hemorrhage into the pelvic tissue.

Ureters and bladder are negative.

Bladder: Is negative.

Stomach: Is negative. Small intestines show a moderate amount of hypertrophy of the solitary follicles. There is no evidence of the greenish tint in these areas.

Bone marrow: Removed from the end of the right femur is chocolate color, semisolid. Sections of the vertebræ show no evidence of tumor in these areas.

Head: There are no tumors found in the skull. On removing the vault of the skull there are found several greenish areas measuring from 1*cm*–3*cm* in length which appear to be confined to the periosteum, and also in places involve the dura. On inspecting the bone of the vaults there are no areas of tumor invading the bone. The right parietal bone shows an area of green discoloration on its inner aspect corresponding to an adjacent green subperiosteal tumor.

Brain: Shows no gross abnormality. There is an increase in fluid.

Diagnosis: Acute myeloblastic leukemia; chloroma.

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AUTOPSY REPORT OF A CASE OF BILATERAL GLIOMA RETINÆ WITH GLIOMATOUS INVOLVEMENT OF THE IRIS.[1]

By Dr. ARNOLD KNAPP, New York.

(With eight illustrations on Text-Plates XX-XXI.)

WHILE the early clinical symptoms of glioma retinæ are well known, some of the later manifestations are not frequently observed, as early enucleation is the usual treatment. At the same time, opportunity for an autopsy is unusual, so that the report of some late clinical features—central perforation of the cornea and gliomatous involvement of the iris—and the autopsy findings seem justified.

The patient was a child, 3 years of age, of Greek parentage, who was brought to us in September, 1921, because the left eye was inflamed. When the child was three months old, a yellow reflex was noted in both eyes, and at six months she was placed in a home for the blind. The child in other respects seemed normal; the other children were all healthy. On examining the eyes, an irregular pink, granulating mass was found in the center of the left cornea (Figure 1). This was regarded as a perforation, with the formation of granulations in a low-grade panophthalmitis, not suspecting an anterior perforation of a glioma. The other eye, of normal size, presented a central opacity in the lens and an exudate in the vitreous. The only peculiar feature was a small, soft swelling under the scalp. The left eye was removed, and on section a glioma of the retina was recognized. While under the anæsthetic, the right eye was more carefully examined, and the mass in the vitreous now suggested a glioma of the retina. Microscopic examination

[1] Presented before the American Ophthalmological Society, Colorado Springs, June, 1923.

ILLUSTRATING DR. KNAPP'S ARTICLE ON "A CASE OF BILATERAL GLIOMA"

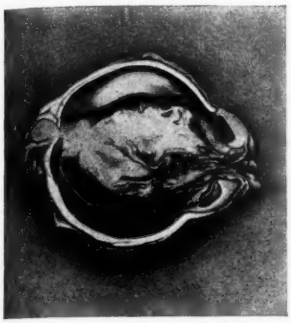

FIG. 1. The left eye showing the corneal perforation.

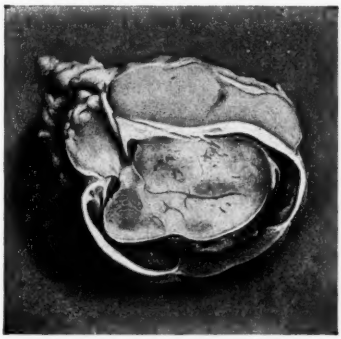

FIG. 3. The right eye.

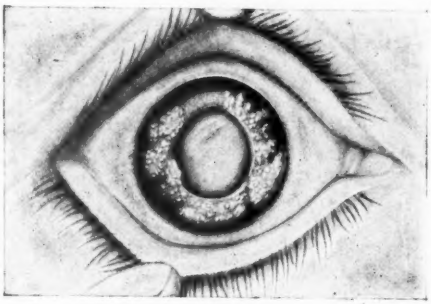

FIG. 2. Gliomatous involvement of the iris.

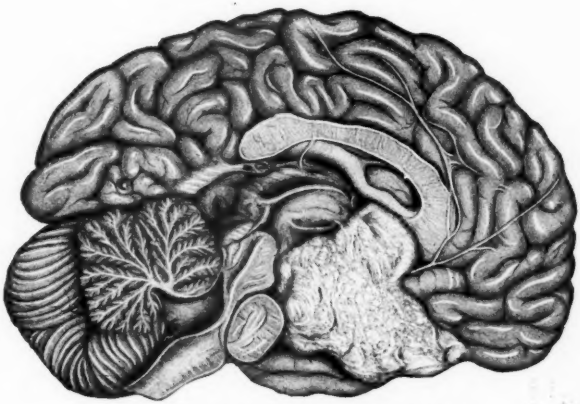

FIG. 4. Glioma of midbrain, direct extension.

ILLUSTRATING DR. KNAPP'S ARTICLE ON "A CASE OF BILATERAL GLIOMA"

FIG. 5. Metastatic extradural tumors on the inner surface of the vault.

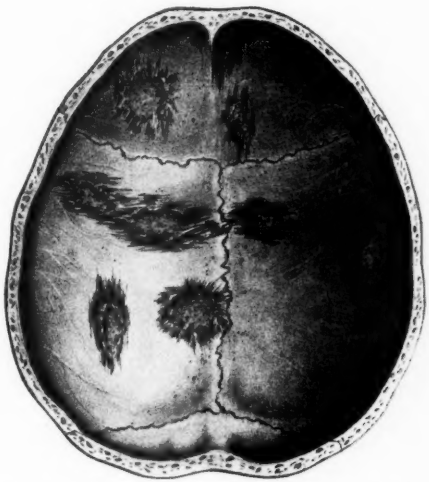

FIG. 6. Bone lesions corresponding to preceding.

FIG. 7. Sub-periosteal tumors of ribs, seen from within the thorax.

FIG. 8. Cross section of rib showing sub-periosteal tumor.

of the left eye showed a central gap in the cornea, filled with gliomatous material, continuous with the principal tumor filling the eyeball. The lens had disappeared. The ciliary body was transformed into a thick, gliomatous structure of well-stained cells which had infiltrated into the stroma of the iris and also extended along its posterior surface. Collections of glioma cells were spread over the posterior surface of the cornea. The choroid was not involved except for a circumscribed area at the equator and in the parts directly adjoining the invaded optic nerve.

The way in which this perforation of the cornea occurred was subsequently demonstrated by the clinical course of the other eye. There was an increasing ectasia of the cornea, with enlargement of the eyeball, corneal ulceration from exposure then developed, followed by perforation. A subsequent stage would be the formation of a vascular, mushroom-like tumor,—the fungus hæmatoides of the older writers.

A local recurrence took place in the left orbit two months later; this was removed; there was no change in the right eye. Six weeks later, December 15th, 1921, the child was again brought to the hospital with another recurrence in the left orbit. The right eye had enlarged, and the iris showed peculiar nodular changes (Figure 2). A vascular mass had pushed through the tissues at the root of the iris, just as if coming from the ciliary body. The iris surface was covered with vascular, round, white nodules, and loose whitish material resembling hypopyon appeared in the bottom of the anterior chamber. Subsequently a hemorrhage in the anterior chamber took place, the iris nodules were no longer defined, the iris markings became lost, and the anterior chamber contained a coagulated mass. As the cornea was not covered, an ulcer from exposure developed in the lower third which perforated, and a purulent-like fluid escaped.

Involvement of the iris in glioma is an unusual occurrence. I have found the following references in the literature without finding a description or illustration resembling this case. The extension of a glioma of the retina to the choroid regularly occurs at the place where the choroid and retina join, namely, at the entrance of the optic nerve, as was originally shown by Herman Knapp in 1868. The lamina vitrea usually prevents the extension at other points (Wintersteiner). On invading the papilla, the tumor progresses into the adjoining choroid,

often producing a characteristic pillow-like infiltration. The tumor finally continues uninterruptedly to the ciliary body and iris. The entire uveal tract may be converted into a tumor mass consisting of connected convoluted nodules, separated by a pigment line from the necrotic retinal tumor in the center of the eyeball. In other cases, as in this, the extension to the anterior part of the eyeball takes place by a gliomatous growth along the inner surface of retina and choroid, extending to the orbiculus ciliaris, the ciliary processes, and the zonula. The ciliary body is overgrown by thick, gliomatous material which penetrates between the ciliary processes, replacing them by a tumor mass, and then infiltrates the iris stroma. In some cases the surface of the iris and the posterior surface of the cornea are covered by small glioma nodules or extensive gliomatous masses, which are probably of similar origin as the isolated nodules in the vitreous. The gliomatous proliferation in the iris may become free in the anterior chamber, or the loose gliomatous cells may sink to the bottom, producing an hypopyon-like deposit that has frequently been mistaken for pus.

In our case the mass in the left orbit increased in size, the glands in the left side of the neck enlarged, and general petechial hemorrhages occurred. The condition of the child failed rapidly without any signs of cerebral involvement, and death took place on January 30th, 1922.

The right eye (Figure 3) was removed for microscopic examination. The eyeball presented a large epibulbar tumor. The optic nerve sheath was completely filled with gliomatous cells. The eyeball was somewhat shrunken and completely disorganized, the interior structures were replaced by a gliomatous mass, chiefly necrotic, with irregular pigmentation and cholesterin crystals. The sclera was infiltrated with masses of glioma cells. The anterior chamber, iris, and ciliary body were transformed into a gliomatous mass, with obliteration of all details. At the center of the collapsed cornea there was a perforation filled with necrotic, gliomatous material. The optic nerve was uniformly enlarged, with a constriction of the optic foramen.

Autopsy was performed five hours later by Professor James Ewing, of Cornell University.

Body of extremely emaciated female, 3 years old.

Left Orbit: Protruding from the left orbit is a tumor mass about 8 x 10*cms* over which the lids are greatly distended but not necrotic. The tumor is soft and lobulated, its surface is infiltrated with blood, but there is no obvious necrosis. The mass occupies the entire orbit and is adherent to the periosteum below, involving the bone of the floor of the orbit and the inner surface of the lower lid. It is free above.

Right Orbit: On reflecting scalp in region of lacrimal gland, a bluish tumor mass is exposed which fills the orbit above. This mass is covered by periosteum and proves to be an extension into the orbit from a subdural tumor which half fills the right middle fossa.

Left parotid gland enlarged.

Thymus: Normal.

Heart: Normal.

Lungs: Normal.

Spleen: Size normal, consistence much increased; very elastic; markings normal; surface shiny.

Liver: Slightly enlarged; presents on its surface as well as on section many miliary to bean-size tumor nodules.

Kidneys: Very firm but show no abnormality.

Pancreas: Normal.

Stomach and Intestine: Normal.

Lymphatics: Mesenteric nodes slightly yellowish and rather prominent. No lymphatic metastases anywhere demonstrable.

Chest: Protruding into the chest cavity beneath the periosteum and replacing (?) the bone substance, over segments two *cms* up to the entire rib are soft, opaque, yellowish tumor masses. Every rib is involved except the two lower ones.

Pelvis: In the left false pelvis there is a tumor mass, size of hen's egg, beneath periosteum of ilium.

Scalp: The periosteum is protruded by very many flat elevations lying beneath the periosteum and rising to about five *mm* above the surface of the skull.

Brain: Beneath the dura and not opposite the subperiosteal tumors, but scattered all over its convexity, are flat, vascular, soft tumor masses, 1–5*mm* thick (Figure 5). These are outlined in a zone of hemorrhage and some have caused erosion of the bone (Figure 6). The brain is adherent to the periosteum at the exit of the optic nerves, and all the tissue around the optic chiasm is converted into a soft tumor mass (Figure 4). This tumor mass has destroyed two-thirds of the optic thalamus.

Anatomical diagnosis: Recurrent glioma retinæ, left

orbit, with metastatic growths in the right orbit, skull, peri-cranium, liver, ribs, and left ilium.

This case, aside from the perforation of the cornea and the peculiar involvement of the iris, illustrates certain striking peculiarities in the course of a glioma of the retina. First, its extension by continuity along the optic nerve to the brain (Figure 4) and to the bony walls of the orbit and the adjoining bones of the face, which explains the tendency of glioma of the retina to local recurrences. Second, as evidences of metastasis, the multiple isolated subperiosteal tumors in the skull. These appeared on the inner surface of the vault of the skull as red, nodular masses, firmly adhering to the dura (Figure 5) and with a corresponding defect in the bone, with an irregular, jagged margin (Figure 6). On the external surface of the skull there were a number of small, yellow, subperiosteal tumors without involvement of the bone. Third, the most noticeable tumors in distant bones occurred in the ribs (Figure 7), with the development of diffuse, nodular subperiosteal tumors in which the bone was not macroscopically involved (Figure 8). Numerous nodules were present in the liver, both superficial and deep. The parotid gland was enlarged.

Metastases in retinal glioma are rare, and these are preferably in the bone and rarely in the internal organs. Sarcoma of the choroid, on the other hand, rarely extends to the orbit, it does not recur locally, and is very prone to metastasize in the liver. It is also curious that the metastases occur about the eye and decrease with the distance away from it. The predilection for bone involvement is striking. This is especially so when their rarity in choroidal tumors is noted. Wintersteiner found in glioma of the retina, tumors of the skull as frequent as brain involvement (40 and 43 respectively), while the liver was involved in only seven; Fuchs found in 33 choroidal sarcomata the metastases present three times in the bones and thirty times in the liver. There seems to be a striking predisposition in the child skeleton to these periosteal metastases.

Metastases in distant organs are unusual. A reason for this is possibly that there are but few and small vessels present in the retina. Glioma cells do not readily penetrate the vessels,

as the vessels in the retinal circulation are small and the walls are thickened at an early stage. It is a matter of experience that if no local recurrence follows enucleation, metastases do not occur. When the choroid is invaded, conditions more favorable for metastases are given. In the early stage, if no local recurrences takes place, no metastases appear. Not until the choroid is invaded do conditions favorable to metastases develop and, through the additional medium of the epibulbar and orbital tumors, opportunity is given for retinal glioma to grow along both blood and lymph vessels.

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THE NEW UNIT OF VISUAL ACUITY AND ITS PRACTICAL USE.

By Prof. L. de BLASKOVICS, Budapest.

(With three figures in the text.)

FOR many years the older unit of 1′ for expressing the visual acuity, has been regarded as unsatisfactory. It has not been proved, and furthermore is always accompanied by the difficulty of expressing the vision in fractions. It gave occasion, also, to mistakes. Many were led to believe that $\frac{10}{10}$ represents "normal" vision, and a smaller fraction, a part thereof. Snellen himself protested against this error which to this day is still used to express "full vision." How could we remedy this fallacy? The error is evidently the result of a failure to consider the inverse ratio of the visual angle to the vision. Therefore it is faulty to consider the visual angle of the unit as of small value. The refraction of light is also measured with a reciprocal value, namely, the focal distance of a lens. Donders eliminated this fault by replacing the inch with the relative greater value of the meter for the unit of the focal distance. Thus the dioptric value of nearly all of the lenses used is designated by a full number. To illustrate:

$R = \frac{1}{F}$. If $F = \frac{m}{n}$ then $R = n$ meter-lens, which means that the record is nearly always a full number.[1] I propose the same procedure for measuring visual acuity. The solving of the question becomes simple and natural if we accept one degree, the unit of goniometry, for the visual angle of the unit of vision. We then find:

[1] In which R = diopters, F = focal distance and n = distance from lens.

$$V = \frac{1}{a}; A = \frac{1^\circ}{a}; \text{ then } V = n.^1$$

In which a = the visual angle, and n = the degrees subtended by the angle.

The visual acuity is indicated by a full number, for a visual angle of 1° is a large value.

I introduced this proposition at a most inopportune time (in 1914).(1) This was perhaps the reason for its remaining unobserved. The new unit was named "Oxyoptre," and for it I proposed the sign °. Referring the Oxyoptre as the "minimum separabile," it is the $\frac{1}{60}$ part of the Snellen unit—there being 60′ to 1°.

The definition of the proposed unit therefore should read: ***The Oxyoptre is the reciprocal value of the visual angle, expressed in degrees.***

Similarly, Snellen's unit could be expressed as, "the reciprocal value of the visual angle expressed in minutes." One Oxyoptre corresponds to the counting of fingers at 1 meter or $\frac{1}{200}$ (Snellen's chart). Visual acuity of a lesser degree can scarcely be measured. According to Landolt, (2) the movement of the eyes is necessary to recognize objects with a greater visual angle than 1°; which then no longer corresponds to the definition of visual acuity. The practical advantages of recording visual acuity in Oxyoptre can be recapitulated as follows:

I. It is superfluous to count in fractions. Every one knows the difficulties encountered. Comparing two values is also difficult at times. For instance the difference between $\frac{4}{4}$ and $\frac{5}{6}$ can be found only by calculation. The difference between 75/$_o$ and 50/$_o$ is immediately seen. One does not see the relationship between counting fingers at 4 meters and $\frac{4}{10}$ but it is immediately noted between 4/$_o$ and 40/$_o$.

II. Counting with small values of vision is more simple when doing it with Oxyoptries.

Henker (3) called our attention to this fact, while occupying himself with the construction of magnifying spectacles for the weak sighted. For undisturbed reading one requires a visual acuity of 30/$_o$ ($\frac{20}{40}$) with a vision of 10/$_o$ ($\frac{20}{120}$) one requires a three times magnification: with a vision of 6/$_o$ a five times

magnification is necessary; that is, $30/_o$ must be divided with the number of the found Oxyoptries one possesses.

III. With the accidental circumstances, that $1/_o$ corresponds to the counting of fingers at 1 meter, we are enabled to determine higher degrees of visual acuity without any test type. My experiences in this direction showed sometimes an astonishing uniformity between the Oxyoptric values and that of counting fingers. (Fingers should be placed directly in front of a dull black surface and in good light.) Of course this method of examination is employed only exceptionally and acts as an auxiliary.

IV. The visual acuity of a healthy person (chiefly in the young) is about $100/_o$. By this happy circumstances it is easy to make the laity understand *e.g.*, in medico-legal cases, when recording in Oxyoptries we need only tell that the number nearly expresses in percentage the visual acuity of a good-sighted young person.

V. The number 60, being greatly divisible, makes every conversion very simple. Nearly all the usual records of the visual acuity can be expressed with a full number, thus, the values of Snellen expressed in Oxyoptries are 6— 10— 15— 20— 30— 40— 60. The international values are 6— 12— 18— 24, etc.

VI. The small unit on the list types makes possible a regular progression mostly in full and round numbers. An arithmetical progression is 5—10—15—20—25, etc. A geometrical progression with (multiplicator) of $\sqrt{2}$ is 5—7—10—15—20—30—40—60, etc. This already is more regular than that of Snellen. The progression with the (multiplicator) $\sqrt[3]{2}$ is approximately 6—8—10—12—15—20—25—30—40—60, etc. In each case the growth of the value is clearly marked.

VII. The visual angle, the real means of measuring, is expressed in the number of oxyoptries just as the focal distance is found by the number of dioptries. The visual angle is simply found by taking the reciprocal value of the oxyoptric number, *i.e.*, the visual angle of $15/_o$ is $\frac{1}{15} = 4'$.

Although the Oxyoptre is a new unit, its application does not necessitate new test types. Nevertheless I designed new ones, striving to answer all the demands. The visual angle of the object is the important factor. As is known, this

question is still unsolved. The recognition of the object is only possible if it recalls a memory image. But this takes place prior to the real recognition. As Kreiker (4) showed, this relative recognition is approximately 130% greater than the "absolute" recognition when the patient is accustomed to that particular chart. Thus it happens, when examining with the same sign, *i.e.*, with Landolt's ring, the patient fails to recognize two or three signs of lesser value and then recognizes the next one of greater value.

So, it frequently happens, that on the international test types, the patient recognizes the 0.7 and fails on the 0.8 and 0.9 but recognizes the 1.0 value well. The 0.7 should in such an instance correspond with the "absolute" while the 1.0 should correspond with the "relative" recognition. In several instances have I seen the memory images play an important rôle in recognition of an object, *e.g.*, a person who observes Landolt's ring for the first time will not recognize it nearly so well as after repeated examinations. I myself recognize the 3*mm* thick ring first from a distance of 15 meters and later from a distance of 18–19 meters. My assistants had similar experiences. But this was not a true recognition. I can prove this by the fact that when experimenting with Snellen's and Landolt's sign, we could frequently tell in which direction both were open, but failed to recognize Snellen's sign from Landolt's ring. The recognition took place obviously, due to the fact that one side of the sign was lighter than the other. The influence of the psychic factor can be demonstrated also by other experiments. It is more difficult to recognize a sign if we do not know whether it is a letter, numeral or other object. The results will be different if we show only letters or letters and numerals mixed, or if some be inverted: again, information given the patient also alters the result of the examination.

All these facts show that only a relative value is recorded. Illiterates and small children recognize the signs with greater difficulty, not being familiar with the ordinary signs, therefore appear to have less vision. Engineers trained to the various geometrical forms, surpass others in recognizing these signs. These observations can frequently be made. The International Ophthalmological Congress of 1909 was therefore

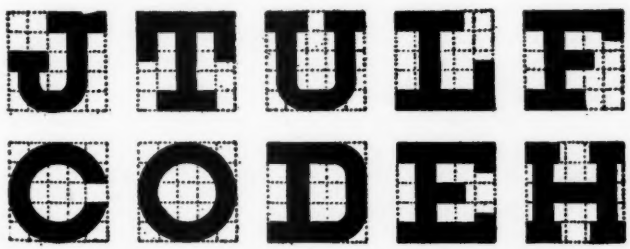
correct in determining a uniform sign for eliciting the visual acuity (5). Although the accepted Landolt's ring fails to give an accurate value, nevertheless it should make the records more uniform throughout the world, if generally accepted. Until the real value is determined, we cannot do any better. The international decree makes the work of the test-type composer simpler. However, there are difficulties, for nobody likes to omit the accustomed letters and numerals which he long has been accustomed to and through which he makes his determinations more quickly and comfortably. Hess (6) solved this problem so that the numerals and letters must be adopted to answer the purpose of Landolt's ring. He solved the problem on the international test type by choosing the easy numerals and made them of such a size that their recognition would be equivalent to the corresponding Landolt's ring. However, by this method a smaller visual angle is apparently the basis of the examination. This fault can perhaps be avoided by permitting the letters to remain in their original form and composition but choosing only those letters whose visual angle equals that of Landolt's ring. I began this choice by drawing the letters from the "antique" alphabet promiscuously on a cardboard. Approaching from a great distance one soon observes that there are no two letters of an equal value. They are recognized one by one from different distances. Nevertheless there are letters in this alphabet whose values approach Landolt's ring. These are (experimenting with twenty-three patients) T.U.C.O. and D. These five letters being insufficient for a variation, I chose those which could to some degree be remodelled without disfiguration for proper recognition. After many experiments I found the following letters useful. T.L.F.E. and H. Frequently and in several ways altering their form in my experiments with 100 normal persons, I finally evolved the letters to conform with the corresponding Landolt's ring approaching the same visual angle. Of course the principle of the "minimum separabile" was sacrificed. However we did not consider that seriously for the real value remains the corresponding Landolt's ring and is calculated from that. Therefore we speak to-day only of the angle of recognition. With Landolt's ring we test strictly the light sense, for the relative

recognition takes place as soon as the patient recognizes the lighter side of the ring. Examining ourselves unbiassed we observed the opening of the ring far sooner than we saw it clearly, *i.e.*, my eye with a visual acuity of 90/$_0$ ($\frac{10}{11}$) observed actually the open ring with its opening only in a value of 50/$_0$.

I must mention that in referring the oxyoptre as the "minimum separabile," it is done so only with reference to the opening of the ring. Although we cannot determine the optical solving power, every object will have some important part measurable with an angle. It is better to apply the angle to the thickness of the letter than the entire letter itself, for a letter could be constructed in other proportions, *e.g.*, 3×5.

In testing vision with letters, eclecticism, surely plays a part in the recognition. The patient while not recognizing it searches for a memory picture resembling it and chooses between the similars. This psychical factor is shown by some interesting facts. The circle is very easy to recognize if it is a numeral, in fact the easiest, as we have only this one round figure with a white center. But if it represents a numeral it is a little more difficult than Landolt's ring evidently because we must choose between more letters. The letter C quite equals with Landolt's ring, is also a little more difficult than Landolt's ring, but easier than O, for the observation of the position of the opening is already the deciding factor: and, on the contrary, the recognition of the absence of the slit as a negative factor, requires a more precise observation.

The chosen and partly remodelled letters are in their new form and in their ranges of recognition:



The dotted lines in which the figures lie are self-explanatory. I shall mention only the essential modifications.

The length of the two down strokes of the letter T is 1½ of the thickness of the letter: (1½ t) similarly with the upward stroke of the letter L. The upper horizontal line is 3½ t.[1] The downward stroke of the letter F is ½ t: its tongue is 1½ t. The opening of the letter H above and below is 1½ t. The first row of letters is more easily recognized than the second row. Between the two lines stands the value of the Landolt's ring. These experiments were performed on the previously mentioned normal sighted persons. Of course, in spite of all labor the letters are not equal. In the past ten years I have been using letters drawn with an exactness of 0.1*mm*, during which time 30 to 40 thousand tests were made. This daily exercise rendered possible continuous improvement. Some of the letters were tried in 10 to 20 various forms. An experienced eye need only glance at these letters and note that there cannot be a great difference between them. In our experience we found that the difference between the upper, lower row and Landolt's ring is a difference of only 5 to 6% This difference is not great, when we consider that with the old method we cannot obtain an exactness closer to 100%. Practically this difference is negligible, for a difference of less than 20% increase between lines is not essential. On the basis of 100 experiments Kreiker found that the letters are on an average 6% easier to recognize than Landolt's ring. Therefore it is always advisable to compare results with Landolt's ring as Hess already has proposed. I attempted avoiding this error in my now published test-types by placing two letters on a line, one being round and the other angular. The first letter being easier, whereas the second (the control) is more difficult than Landolt's ring. The value is recorded if the patient recognizes both letters on that line. Naturally a comparison with Landolt's ring would be more advisable. For those adhering to the values of Snellen's chart, I constructed a test-type with numerals which are equal in value to Snellen's chart as Kreiker mentioned. These numerals are in their range of values as shown on following page.

We were obliged to change Snellen's construction to have corresponding values. The figure 1 received a base, the length

[1] "t" = the thickness of the upright stroke of a letter.

of 4a[1]; the obliquity of the 7 was diminished and received a stroke through the body. The first stroke of the 4 was made more perpendicular. The curve in the forefront of the 2 was

lessened; in the back it received a little ($\frac{1}{4}$a) addition. The opening of the 3 is $1\frac{1}{2}$a; the tongue ending $1\frac{1}{2}$a distant from the opening. The values of the numerals approached each other perhaps closer, as they could be better modelled, than the other numerals and Landolt's ring. Therefore they occur in almost all values. The five letters proved sufficient to secure the necessary attention, for they are read together and the changed numerals make another impression, *e.g.*, 42 and 24. The 4 and 2 are very near to the corresponding Landolt's ring. Therefore in the two values both are employed. For testing persons who cannot read but know numerals the Snellen–v. Amon sign is recommended. As previously mentioned illiterates are less skilful in recognizing signs; therefore we are nearer to the real visual acuity with these people when testing them with numerals of V. Amon's sign. Landolt is correct in rejecting the testing of vision by counting fingers and proposing the use of his ring at various distances. For such an examination my test-type contains Landolt's ring and Snellen–v. Amon's sign on separate charts. Their value is $5/_{o}$ in a distance of 5 meters. In making the examination with this test-type the distance of recognition expressed in meters gives the visual acuity oxyoptres. Thus the tables give values from 0 to $120/_{o}$ ($\frac{5}{120}$) in a progressive manner. I recommend the record V. = 0 for cases without light perception. If light perception is elicited it is recorded serviceably with relative 0 or $\frac{1}{120}/_{o}$. The size of a visual object $2 \operatorname{tg} \frac{\alpha}{2}$. As we have $2 \operatorname{tg} 90/_{o} = \infty$, this record corresponds with $\frac{1}{\infty}$ and designates endless great objects or light and dark. I do not recommend the use of the signs in oblique directions. On my test-types the ring and v. Amon's sign

[1] "a" = the value of one small square, or subdivision of the large square.

	/o	*V in decimals*	$V = \frac{a}{D}$	$v = \frac{I'}{a}$	
1m.	1	Fingers in 1m.		$\frac{1}{60}$	1m.
2m.	2	" " 2m.		$\frac{1}{30}$	2m.
3m.	3	" " 3m.		$\frac{1}{20}$	3m.
4m.	4	" " 4m.		$\frac{1}{15}$	4m.
5m.	5	" " 5m.		$\frac{1}{12}$	5m.
	6	0·1	$\frac{10}{100}$	$\frac{1}{10}$	
	8	$0{\cdot}1\dot{3}$	$\frac{10}{120}$	$\frac{1}{7.5}$	
	10	$0{\cdot}1\dot{6}$	$\frac{10}{120}$	$\frac{1}{6}$	
	12	0·2	$\frac{10}{100}$	$\frac{1}{5}$	
	15	0·25	$\frac{10}{40}$	$\frac{1}{4}$	
	20	$0{\cdot}\dot{3}$	$\frac{10}{30}$	$\frac{1}{3}$	
	25	0·4 <	$\frac{10}{25}$	$\frac{1}{2.4}$	
	30	0·5	$\frac{10}{20}$	$\frac{1}{2}$	
	40	$0{\cdot}\dot{6}$	$\frac{10}{15}$	$\frac{1}{1.5}$	
	50	0·8 <	$\frac{10}{12}$	$\frac{1}{1.2}$	
	60	1·0	$\frac{10}{10}$	$\frac{1}{1}$	

Tab. Ia

Tab. Ib

Tab. II

Tab. IV

← visions > 60/o →

80	100	120	v. in oxyoptres.	$v = \frac{a}{\delta}$	$\frac{10}{16}$	$\frac{10}{12}$	$\frac{10}{10}$
$1{\cdot}\dot{3}$	$1{\cdot}\dot{6}$	2·0	v. in decimals	$v = \frac{I'}{a}$	$\frac{1}{0.75}$	$\frac{1}{0.8}$	$\frac{1}{0.5}$

are only in the four principal directions. The illiterate chart is also useful in forming the patient as to the course of the examination. On the back of these charts are found the tables of exchange. All the values are shown in the following illustration, together with the tables of exchange.

This illustration gives at the same time the true reproduction of the test-types I a, I B, II–IV diminished to ½. Chart III contains the ring which is to be used in conjunction with the letters. Chart V shows the Snellen–v. Amon sign. If we make a survey of the values we note that the range of oxyoptres is clearer than the record in decimals or in Snellen's fractions. In the last line vision is expressed with a fraction containing the numerator 1. This corresponds with the formula $v = \frac{1}{a}$. The denominator shows the visual angle in minutes. This is clearer than multiplying the numerator and denominator with the distance of testing. This is superfluous because the examination is made at a distance which eliminates the accommodation. It is of greater importance to note the figures or letters used. We could write V sn (vision with Snellen's test-type); V i (international test-type); Vo (ring); Ve (illiterates); Vn (numerals), etc.

In making known my test-types I have tried to show the practical use of the oxyoptre, the acceptance of which should stop some present contradictions and much annoyance.

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REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London, and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

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(*Continued from July issue.*)

IX.—THE CONJUNCTIVA, CORNEA AND SCLERA.

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BEDELL'S (137, **So-called solid œdema**) case was characterized by a broad flat pericorneal mass which extended more than half way around the limbus. It lay under the conjunctiva and was œdematous in appearance. On being excised it proved to be a true lymphoma.

The peculiarity of the case reported by BERGMEISTER (138, **Epibulbar tuberculosis**) was that the starting point of the tuberculous disease was in the subconjunctival and episcleral tissue. Thence it spread along the anterior ciliary vessels into the globe itself, and also into the limbus in the region of Schlemm's canal.

CLAUSEN (139, **Case of typical vernal catarrh treated with afenil**) obtained distinct subjective improvement after one intravenous injection of 10 *ccm* of afenil. After a second injection on the next day there was no improvement. A third injection four days later caused only a slight transient improvement. The palpebral changes were not affected at all, while the growths about the limbus were distinctly flattened,

possibly as a consequence of the cooling of the atmosphere. Two injections of afenil produced no result in a patient with severe hay fever, and just as little benefit was obtained in scrofulous keratoconjunctivitis.

EGTERMEYER (140, **Afenil in vernal catarrh and hay fever conjunctivitis**) reports four cases of vernal catarrh treated as suggested by Cords with three intravenous injections of afenil, a preparation of calcium chloride and urea, without obtaining any subjective or objective change in the clinical picture. Beneficial effects were obtained in four cases of hay fever conjunctivitis. In two the attacks were completely checked after a single injection; one patient required two injections, but the fourth was apparently much improved for a fortnight after three injections and then suffered a relapse.

PICHLER (150, **Afenil in vernal catarrh**) thinks afenil a valuable addition to our armamentarium, but one from which too much should not be expected, it must always be used in connection with the usual local treatment.

WACHTLER (160, **Treatment of vernal catarrh with afenil**) contributes a case in which after two intravenous injections of 10 *ccm* of afenil the objective and subjective signs of vernal catarrh with well-developed palpebral changes disappeared completely.

Whether there are phlyctenulæ without tuberculous infection is a question answered differently by different writers, although the question most discussed to-day is whether the phlyctenulæ are caused by toxines, or through the influence of dead or living bacilli. According to Kleinschmidt (145, **Are there phlyctenulæ without tuberculous infection**) a small portion of the children with phlyctenular conjunctivitis do not react to tuberculin and are free from tuberculosis.

STARGARDT (154, **Phlyctenulæ of the palpebral conjunctiva**) saw well-marked phlyctenulæ on the palpebral conjunctiva of a patient, 24 years old, who presented the typical picture of a severe bilateral phlyctenular conjunctivitis.

LINDNER (147, **Topography of parasitic conjunctival germs**) says that gonococci are to be found in the epithelium of the conjunctiva before they appear in the secretion, so that a microscopical examination of a bit of this epithelium taken from a suspicious case can make the diagnosis of gonorrheal

conjunctivitis positive in the first hours of this disease. Phagocytes remove the Koch-Weeks bacillus earlier and more thoroughly from the deeper epithelia than any other germ; these germs are therefore found only superficially and for a short time in the epithelium. The influenza bacillus is a parasite whose topography is similar to that of the Koch-Weeks bacillus; it is perhaps the only germ which is frequently found in greater numbers in the secretion than in the epithelium soon after the beginning of the inflammation. The pneumococcus is a typical parasite of the normal conjunctiva; its preference for the bulbar conjunctiva is just as marked as that of the Koch-Weeks bacillus. As regards the micrococcus catarrhalis and the meningococcus as provocative agents of conjunctivitis, Lindner has found no gram negative diplococcus, aside from the gonococcus, proliferating in the normal epithelium. The diphtheria bacillus proliferates in patches on the epithelial cells and penetrates into the epithelium. In fresh acute cases of trachoma one finds inclusions and free initial bodies throughout the entire conjunctiva. The Morax-Axenfeld diplobacillus does not grow at all on the normal epithelium of the conjunctiva, but on moist horny epidermis cells, or dead conjunctival epithelium, and in mucous secretion, hence it proliferates at the zone of junction of the epidermis and epithelium. All germs known as surely infectious to the conjunctiva are primarily epithelial parasites.

PILLAT (151, **Topography of the saprophytic germs of the human conjunctiva**) understands as saprophytic germs such as are to be found on conjunctivæ which appear clinically to be perfectly normal. These are, aside from casuals, the xerosis bacillus, the staphylococcus, and the pneumostreptococcus. They are as typically parasitic as the pathogenous germs. Although they grow only on normal living cells, they derive their nutriment from more or less degenerated cell material. Xerosis bacilli were found in 100 per cent. of the eyes examined; on the margins of the lids and in the canthi in 97 per cent., one the palpebral conjunctiva in 82 per cent., on the bulbar conjunctiva in 81 per cent. Staphylococci were found in almost 94 per cent. of the cases, on the lid margins in 72 per cent., on the palpebral conjunctiva in 66 per cent., and on the bulbar conjunctiva in 62 per cent. It may be mentioned that staphy-

lococci are never met with like tufts on the epithelial cells on the normal conjunctiva, but always singly. Pneumostreptococci, found in 40 per cent., do not seem to occur on the margins of the lids or in the canthi; in twelve out of thirteen cases they were found on the bulbar conjunctiva and once on the palpebral. Diplobacilli were found in 22 per cent., about equally common on the margins of the lids and the palpebral conjunctiva; in two out of seven cases they occurred on the bulbar conjunctiva. Culturally xerosis colonies were obtained from all six cases investigated, staphylococcus albus non liquefaciens likewise in 100 per cent. Staphylococcus pyogenes aureus was cultured once from the palpebral conjunctiva, sarcina flava once from the puter canthus.

LINDNER (148, **Blennorrhea neonatorum**) says that out of the great number of cases of this disease seen by him he has not seen one that surely began after the fifth day. Careful studies show that the blennorrheas which set in later are, without exception, inclusion blennorrheas. An inclusion blennorrhea begins at the earliest on the fifth, commonly on the seventh or eighth day after birth, and can set in just as stormily as the gonorrheal disease. Crede's method does not protect against this form of blennorrhea. The virus comes, like the gonococcus, from the genitals of the mother, is transferred to the conjunctiva of the infant during birth, and also occurs in the male urethra. The identity of the virus with that of trachoma is disputed, but when it is placed on the conjunctiva of adults it gives rise to clinical pictures which are difficult to differentiate from mild cases of trachoma.

STEINER (155, **Trachoma**) describes spots of pigment on the tarsal mucous membrane as a little known complication of trachoma which is often to be found among the Javanese and other colored races. Another complication of the same disease is the development of large tumors of the lid through amyloid degeneration of the conjunctiva and tarsus.

PATTON and GIFFORD (149, **Agricultural conjunctivitis**) report six cases of severe conjunctivitis characterized by extensive swelling of the lids involvement of the preauricular, cervical glands, ulceration of the skin and a dense false membrane on the conjunctiva. The cornea was more or less involved in ulceration. All the patients were engaged in agri-

cultural pursuits. None had been exposed to diphtheria. The staphylococcus and bacillus xerosis were found in all cases. A large gram positive anærobic bacillus was present in all but one case and was probably the cause of the disease. They suggest that the condition be called "agricultural conjunctivitis."

STOCK (156, **Cornification of the conjunctival epithelium**) saw two cases of this very rare disease. Both patients came from the same place, so perhaps the same cause was present in both. One patient was a man 53 years old; to the outer side and below the limbus was a place about 1½*mm* square which was somewhat protuberant and could be moved with difficulty over the subjacent tissue. The second patient, a man 50 years old, presented a similar growth some 2*mm* square at the inner margin of the limbus and extending into the clear cornea. In both cases the tumor was removed and the galvanocautery applied to the base. Examination showed in each case an extensive cornification of the surface of the epithelium with a considerable deposit of amyloid. In one case a reactive inflammation was present in some places. It is noteworthy that all cases hitherto observed appeared in the palpebral fissure.

ZELLER (161, **Studies of the conjunctival vessels**) finds that many conditions of the vessels, especially of those in the palpebral fissure, must be accounted for with care, as these are far more exposed to external influences than the vessels anywhere else in the body. But yet, with certain limitations, conclusions can be drawn from the changes in the conjunctival blood vessels as to vascular and general diseases, and particularly as to local eye diseases. He found the rapidity of the current of red blood cells in arteries from 0.01 to 0.03*mm* in diameter to be an average of from 1.6 to 3.5*mm* per second, in veins 0/7 to 1.8*mm* per second. In old persons the current is slower than in young ones. He could not find a constant ratio between the rapidity of the current and the diameter of the vessel. Miliary aneurysms may be caused by vascular and general diseases, but also occur in cataract, synchysis scintillans, syphilis, gout, diabetes, nephritis and cachectic conditions. That the recognition of such changes may have practical importance is shown by two cataract patients who

had numerous dilatations with slight signs of stasis in the capillaries of the limbus, and were subsequently found to have fluid vitreous. A stretching of the vessels is usually characteristic of sclerosis; this causes an increase of the physiological curves into little loops. Aneurysms, which seem to prefer the places of division of the vessels, and varices are very common. Syphilitic diseases of the vessels frequently show themselves only in spindle shaped and saclike dilatations particularly of the capillary loops and the venous portion of the smallest vessels. A typical condition of the capillaries in diabetes is a dilatation of the connecting part between the arterial and venous portions of the vessel, and usually of the venous trunk itself.

According to FREY (141, **Sensitiveness of the human cornea and conjunctiva**) the cornea and conjunctiva are sensitive to pain and cold exclusively, while sensations of touch and heat are lacking. In the cornea there are only two different sorts of nerve endings, one chiefly in the deeper layers of the corneal periphery, which may transmit the sensation of cold, while the intraepithelial nerve endings serve the sensation of pain. As regards the faulty localization of pain by the cornea, he has found that an irritation affecting one quadrant of the cornea is commonly localized in another quadrant.

UHTHOFF (157, **Degenerative changes of the cornea and conjunctiva**) describes first a case of dystrophia corneæ epithelialis (Fuchs) with the following findings: The superficial epithelial layer swollen and hyaline, scarcely a nucleus recognizable in the epithelial cells; Bowman's membrane almost totally destroyed; normal corneal lamellæ not to be seen; but replaced by a fine network of confused fibres; in some places a transition of the swollen, greatly changed lamellæ into the finely fibrillar detritus-like confused network of fibres was to be observed. No signs of inflammation were present in the layers examined. His second case was one of a chronic, deep, circumscribed keratitis with the following findings: The corneal lamellæ were much loosened up, undergoing fibrillary degeneration, and permeated with hollow spaces containing fluid, hence were very œdematous. The impression was given of a chronic inflammatory process with partial transformation of the corneal lamellæ into young bascular connective tissue.

No signs of tuberculosis or syphilis were present either clinically or anatomically. The clinical picture of his third case, one of a deep, circumscribed keratitis which ran an extremely chronic course, is very rare. All treatment, including irradiation, was in vain. No general or local cause could be detected, and the other eye was not affected. Microscopical examination of an abscised portion showed a chronic interstitial inflammation with distinctly beginning development of connective tissue, new formation of vessels, and a marked loosening up and degeneration of the superficial layers of the cornea. The fourth case was one of a bilateral, fairly symmetrical degenerative opacity of the cornea which had gradually extended in the course of many years without material symptoms of inflammation. Aside from slight signs in places of subepithelial chronic inflammation, the corneal changes presented the signs of a tissue degeneration with hyaline swelling in places and the formation of numberless spaces filled with fluid. Finally he described the anatomical changes in the palpebral conjunctiva after a vernal catarrh that had lasted thirty-five years. Clinically the conjunctiva of the upper lid, especially the conjunctiva tarsi, was uniformly thickened and of a pale brownish red color, studded strikingly with numerous little concrements of lime so as to present a whitish, granular appearance. The thickening proved to be a uniform proliferation of epithelium which extended deeply in the form of a dense network. Connective tissue with a new formation of vessels was found in the meshes. Numerous concrements, some of them quite large, were deposited in the larger cavities and permeated the entire thickened conjunctiva.

VERHOEFF and FRIEDENWALD (158, **Injury to cornea and conjunctiva due to fish bile**) relate the case of a fisherman who got some codfish bile in his eye and suffered from an abrasion covering about one-half his cornea. It healed leaving an opacity and a false pterygium. Experiments on rabbits showed that fresh fish bile or prepared ox bile will erode the cornea.

BACHSTEZ (136, **Fatty degeneration of the cornea**) reports a case of cholesterine degeneration of the cornea in a patient 36 years old. The process had advanced slowly in both eyes with only slight signs of irritation, and histologically there

were almost no signs of inflammation. The patient had an enlarged thyroid, and the writer discusses the possible endocrine origin of the degeneration.

FRIEDE (142, **Hydroa vacciniforme of the eye**) reports a case of this nature met with in a patient 48 years old. The disease began in early childhood and since the 35th year both eyes had been affected. The condition when seen is thus briefly described: Severe cicatricial changes in the parts of the body exposed to the sun. Right eye: Marked exophthalmos, cicatrices of the skin of the lid, blepharitis, absence of cilia, slight ectropion of the lower lid, mobility of the eyeball limited uniformly in all directions. In the palpebral fissure two porcelain white necroses of the sclera which are sharply defined from the dark red, œdematous surroundings. The entire bulbar conjunctiva adherent and anæsthetic. Cornea smooth, lustrous, anæsthetic; near the scleral necroses a sclerotizing keratitis. Tension low. Papilla distinctly pale. Vision $\frac{6}{36}$. Moderate concentric contraction of the field for white, greater for color; absolute central scotoma for all colors, and a ring scotoma. Left eye: Condition of the lid same as that of the right; bulbar conjunctiva pale, anæsthetic, without vessels in the palpebral fissure, adherent to the sclera, firmly attached over the scleral necroses. In the temporal part of the palpebral fissure a flat staphyloma of the sclera *1cm* in diameter. Cornea, papilla, and tension like the right. Vision $\frac{6}{12}$, $\frac{6}{6}$ with correction. Limits of the visual field normal for white, concentrically contracted for colors, ring scotoma, no central scotoma. Wassermann's, Sachs's, Georgi's and Meinecke's reactions negative. Cryptorchism, sparsely developed hair, no hair in either axilla. It is interesting to note that the eyes did not suffer during the first decade of the disease, but first became affected in later life.

The interesting point in the case reported by GEIS (143, **Acute parenchymatous keratitis during mumps**) is the sudden onset of the keratitis with no signs of cyclitis in a 10-year old girl, and its acute course, run within six days.

One case described by HIPPEL (144, **Pathological anatomy of rare eye diseases**) was that of a woman 68 years old who had suffered from a diffuse episcleritis or scleritis of the entire clinically visible anterior segment of the eyeball, and a disease

of the uvea on the nasal side which probably caused a detachment of the retina that soon became total. The eye was enucleated on account of pain. He found a high grade inflammation of the entire episcleral tissue extending to the optic nerve. The severe inflammatory infiltration of the entire uveal tract with the exception of the iris presented a considable similarity to many cases of sympathetic uveitis. The infiltration extended outward along the posterior ciliary vessels and nerves, involving the orbital tissues, as seen in cases of tumor, tuberculosis and sympathetic inflammation.

KOYANAGI'S (146, **Epibulbar carcinoma appearing like a corneal ulcer**) patient, a farmer 53 years old, had a corneal ulcer which constantly grew larger. It had been in this form from the first with no tumor formation in its shape. The true diagnosis was first made from the histological examination.

SATTLER (152, **Corneal fistula apparently caused by perforation by a partially calcified lens**) reports the case of a woman 42 years old, one of whose eyes had been blinded by an injury twenty-eight years before, who suddenly developed an inflammation in that eye accompanied by severe pain. When examined six weeks later the sharp point of a shrunken, calcified lens occupied a perforation in the cornea. There were no signs of a recent ulcer or of a fresh injury. Anatomically he found a corneal fistula in which lay a calcareous projection from a partially absorbed lens. The corneal epithelium had passed through the fistula into the sac of the lenticular capsule and had overgrown partially the remains of the lens. Probably the injury during youth had caused traumatic cataract with incarceration of the lens capsule in the corneal wound, together with extensive changes within the eye. The remains of the lens could not become completely absorbed because of the incarceration of the capsule and part of it became calcareous with its apex in the anterior chamber.

SCHNEIDER (153, **Pulsating cornea**) saw two cases within a short time. One was of a typical keratoconus with pulsations where the cornea was thinnest, the other followed penetration by a splinter of iron. The latter is said to be the first case on record of a pulsating cornea not associated with keratoconus.

VON DER HEYDT (159, **Clinical observations of the cornea**)

records his observations of lesions in the anterior segment of the eye making use of the slit lamp and corneal microscope. The masses of leucocytes on Descemet's membrane in uveitis he believes to be deposited as droplets and not be formed by the accumulation of the cells, which accords with the opinion of Fuchs. These deposits on the surface of the cornea are sometimes pigmented in the center or covered by pigment. He has examined seven cases of keratoconus and speaks of seeing rings of pigment in the basal epithelium which have been described by Fleischer. In a case of herpes zoster he found a grayish infiltration of about a quarter of the cornea. The surface epithelium over the area was stippled showing epithelial cell erosions and dew-like changes all over the area involved. Descemet's membrane back of this lesion was folded and studded with pigment.

X.—THE LENS.

162. BURDON-COOPER, J. **The ætiology of cataract.** *Doyne Memorial Lecture*, 1922.

163. HESSE, R. **The nature of Vossius' opacity of the lens.** *Zeitschr. f. Augenheilkunde*, xlvi., 1921.

164. LENT, E. J., and LYON, MARTHA B. **Persistence of embryonic fibrovascular sheath of the crystalline lens.** *American Journal of Ophthalmology*, September, 1922.

165. LIPPMANN, W. **Results of operations on diabetic cataract.** *Med. Klin.*, 1921, p. 1115.

166. SCHOEPPE, HEINRICH. **Refractometric studies of the biological behavior of the serum of persons with cataract.** *Archiv f. Ophthalmologie*, cv., 1921.

167. THOMSON, E. **A clinical account of a series of cases of capsular cataract associated with the deposit of pigment at the center of the capsule.** *British Journal of Ophthalmology*, June, 1922.

168. VERHOEFF, F. H., and LEMOINE, A. N. **Hypersensitiveness to lens protein. Patient desensitized and successfully operated on for immature cataract.** *American Journal of Ophthalmology*, September, 1922.

169. WEIDLER, W. B. **Concerning congenital coloboma of the lens.** *Ibid.*, June, 1922.

HESSE (163, **Vossius' opacity of the lens**) reports four new cases, including one in which there was no external traumatism, but a hemorrhage into the anterior chamber from some other cause. From this case it may be concluded that the

essential factor in the production of this condition is not a contusion, but the presence of blood in the anterior chamber.

LIPPMANN (165, **Results of operations on diabetic cataract**) gives the following figures concerning sixty-three patients. Thirty-three were men, thirty women; fifty-three were over forty years old, ten were under; ten were Jews. About six per cent. presented complications, among which were one staphylococcal infection with loss of the eye, one amycotic iridocyclitis with the same result, two cases of iridocyclitis resulting in occlusion of the pupil, five of iritis which did not influence the result as regards vision, one of retrobulbar neuritis immediately after the healing of the wound, one of retrobulbar neuritis which appeared one year afterward, one of diabetic retinitis, six of hemorrhage into the anterior chamber, two of detachment of the retina, and one of secondary glaucoma two years after the operation. On account of the danger of diabetic coma the operation should be performed only when the urine is free from acetone. The diagnosis and prognosis must be guarded because of the possible presence of retrobulbar neuritis, or of diabetic retinitis, which are not demonstrable by the functional test of cataractous eyes.

SCHOEPPE (166, **Refractometric studies of the biological behavior of the serum of persons with cataract**) find no difference in the biological behavior of the serum to the lens albumin between persons who have and those who have not senile cataract, and that a removal of lens albumin cannot be accomplished by the serum from persons who have cataract, or by that from those who have not.

VERHOEFF and LEMOINE (168, **Hypersensitiveness to lens protein**) maintain that about eight per cent. of individuals are hypersensitive to lens protein. If, after operation upon such persons, lens matter is left in the anterior chamber it will cause irritation and inflammation. He believes that sensitive individuals may be desensitized so that this complication may be avoided. He relates a case in which it was shown that injections of pig lens protein controlled the reactions from lens matter left in the eye.

Cataract may be associated with a number of widely different pathological conditions which must be taken into account quite as much as the lenticular condition itself. Among the

commoner causes known as producing opacity in the lens there are traumatisms, direct or in the vicinity of the lens, heat, cold, chemical poisons, electric discharges, light, errors of refraction, general diseases as diabetes, ingestion of poisons, changes in the ductless glands, autotoxæmias and heredity. Cataract may occur spontaneously and, though generally considered to be a senile degeneration, may be a secondary disease as all pathological states of the eye may produce it. The present knowledge of the nutrition of the lens is meager and fragmentary. It is generally held that the nutritive fluid is supplied by the ciliary body and partially by the ciliary processes. After considering the properties and composition of the aqueous fluid and the possibilities of penetration by and imbibition of nutritive fluids by the lens BURDON-COOPER (162, **The ætiology of cataract**) summarizes as follows:—"The food of the lens is secured by it through a modified endosmosis of the intraocular fluid, a molecular imbibition and in all probability a specific affinity of its protoplasm. Disturbance of the nutrition is the initial factor and cause of cataract. When the nutrition is interfered with deleterious substances obtain access to its protoplasm resulting in secondary chemical changes the products of which constitute the opacity." The process of sclerosis of the lens with the associated slight amber coloring consists in the transformation of the lens protein from soluble to insoluble albuminoid, the pigmentation being due to the oxidation of tyrosin, the amino-acid being split off from the albumen molecule, probably by a very slow hydrolysis and oxidised by an oxidase in the lens. This pigmentation associated with sclerosis is more pronounced in Indian lenses than in European ones and Burdon-Cooper thinks that the difference in color is due to the influence of light and heat on the activity of the lental oxidase—this action being protective in character. Burdon-Cooper throws doubts upon the prevalent doctrine that the changes in the lens, spoken of as senile cataract, are accelerated senile changes. Cataractous lenses are usually lighter than healthy lenses, further certain chemical bodies—cholesterin leucin and fat—are found in excess. The presence of such favor the view that they have been occasioned by the transformation of the albumen of the normal lens due to some failure of

nutrition. Burdon-Cooper puts forward his hydrolysis theory. Briefly described it is that a simple decomposition occurs resulting from the assimilation by the proteid molecule of the lens of the constituents of a molecule of water with the production of new substances. The detection of tyrosin, one of the cleavage products in the aqueous after needling, its presence in the aqueous and lens in senile cataract and the diminished weight of the cataractous lens support this theory. Regarding the pathogenesis of senile cataract Becker considers that the prime cause is sclerosis of the nucleus and that the water given up by the sclerosing process clouds the lens by swelling its fibers. It is true that commencing cataractous lenses are lighter and smaller than clear lenses of the same age but in early cataract the lens increases in volume. Senile cataract has been attributed to increased accommodative effort. Burdon-Cooper thinks that hypermetropia and in particular oblique hypermetropic astigmatism is a factor in the production of cataract and produces figures in support of this view. It has been suggested that owing to changes in the epithelium of the ciliary processes and body there may be an alteration in the osmotic pressure of the aqueous and in this way the nutrition of the lens would suffer.

General vascular sclerosis and renal inefficiency have been from time to time considered as the underlying cause of cataract. Though hypertension is not usually associated with cataract Burdon-Cooper thinks that there is usually a renal inefficiency in senile cataract because in cases in which senile cataract is associated with renal inefficiency the lens is shown to have an increase of tyrosin and further that there is a marked similarity in the variation from the normal between the molecular concentration and surface tension of the aqueous and similar constants of the urine. Ultra-violet light has its adherents. Heredity definitely plays a part in the production of cataract but in what way is as yet uncertain. Against the theory that senile cataract is an abiotrophy or premature death Burdon-Cooper points out that it is the oldest part of the lens—the nucleus—which is the least affected in this condition. It would appear that once senile involution has begun regulative devices must intervene to prevent the formation of noxious metabolic products; if these fail there may

appear among the products such as would injure the lens. Regressive changes in metabolism and senile cataract are invariably associated. Burdon-Cooper holds that cataract is the expression of some general metabolic disturbance rather than a local disease. Burdon-Cooper considers that the cataract occurring in young diabetics is a definite entity. Hess thinks that diabetic cataract is due to changes in the iris and ciliary body, others that the lens changes are only indirectly due to the diabetic condition. It is not due to sugar concentration in the aqueous as in cases where the sugar was as high as 10 per cent. in the urine the sugar in the aqueous was never above 0.5 per cent. In addition sugar has been found in healthy lens in experimental pancreatic diabetes. There is an increased amount of cholesterin in diabetic cataract which he suggests is a reduction product of a previously formed acid. But at present all ideas as to the ætiology of diabetic cataract are immature. Intestinal toxæmia appears to be often associated with developing cataract and in 100 cases of incipient cataract Burdon-Cooper found a well-marked indicanuria in 80. Cataract following ergot poisoning takes 3 to 12 months to ripen and then appears similar to the senile form. It has been attributed to spasm of the vessels and consequent interference with nutrition. Cataract occurring in tetany and convulsions seems at present to be due to a toxæmia which is not controlled by a sufficient thyroid and parathyroid secretion. Naphthalene cataract can only be produced when the naphthalene is taken by mouth. How it acts is unknown but it appears to cause the lens to absorb water. Burdon-Cooper points out that its form closely resembles that of the subcapsular senile type of cataract. Cataract due to light and heat has attracted much attention lately. It has been shown that it is the absorption of rays between 11000–1300 A units and those lying between 3000–3500 in the infra red and ultra violet respectively and their conversion into heat in the lens which causes the cataract. With regard to the transient concussion changes in the lens Burdon-Cooper thinks that these are due to alteration in the composition and vital properties of the aqueous so disturbing temporarily the nutrition of the lens.

P. G. Doyne.

THOMSON (167, **A clinical account of a series of cases of capsular cataract associated with the deposit of pigment at the center of the capsule**) gives an account of seven cases, in which there was a deposit of pigment in the center of the lens capsule together with small white opacities situated in the anterior lens capsule or in the extreme anterior layers of the lens. These seven cases were found among seven thousand school children who were undergoing routine ophthalmological examination. This condition was bilateral in three of the cases. The pigment deposit was of the shape of a small more or less complete circle and the white opacities were usually situated just outside the pigment deposit. In two cases of the seven there were adhesions of the iris to the central pigmented deposit. In no cases was there any known history of inflammation of the eyes. It appeared likely that the condition might be due to a pre-natal inflammation which caused a persistence of the fragments of the pupillary membrane. Thomson suggests however that possibly the condition can be explained entirely on the grounds of a congenital anomaly without presupposing pre-natal inflammation. He quotes from an article of COSMETTATOS of ATHENS in which it is stated that membranous vestigia of the pupillary membrane may start from the actual pupillary border and simulate posterior synechiæ. The fact that seven cases out of seven thousand presented this appearance, indicates that the condition is not so very uncommon.

After reviewing the literature LENT and LYON (164, **Persistence of embryonic fibrovascular sheath of the crystalline lens**) report a case of thread-like lines covering the posterior surface of the lens. In and surrounding the meshwork were small round spots. There was no remnant of hyaloid artery. A second case was described as showing an opaque fibrovascular sheath with minute vessels radiating from it. This condition should not be mistaken for glioma or cysticercus.

WEIDLER (169, **Congenital coloboma of the lens**) has found but 33 recorded cases of this defect. His case is unique in that it was not associated with coloboma of the choroid or iris but with microcornea. The lens was notched on the nasal side in the horizontal line.

XI.—THE UVEAL TRACT AND SYMPATHETIC OPHTHALMIA

170. BARTOK. **Sympathetic ophthalmia of long duration.** *Zeitschrift f. Augenheilkunde*, xlv., p. 326.

171. BEHR, C. **Abduction phenomenon of the pupil.** *Klin. Monatsbl. f. Augenheilkunde*, lxvii., 1921.

172. BLIEDUNG, C. **A spontaneous intraepithelial cyst of the iris.** *Ibid.*, lxvii., 1921.

173. DREYFUSS, G. L. **Prognostic guides in isolated syphilitic disturbances of the pupils.** *Med. Klin.*, 1921, No. 51.

174. FUCHS, A. **Sympathetic inflammation and sarcoma in the same eye.** *Klin. Monatsbl. f. Augenheilkunde*, lxvii., p. 303.

175. FUESTI-MOLNAR. **A case of sympathetic ophthalmia after trephining.** *Zeitschr. f. Augenheilkunde*, xlv., p. 326.

176. GILBERT. **Benign tuberculous meningitis in choroiditis.** *Muench. med. Wochenschrift*, 1921, p. 439.

177. GUIST, G. **The behavior of light and dark irides to homatropine and atropine.** *Wiener med. Wochenschrift*, 1921, No. 24.

178. HANKE. **Septic metastases in the choroid.** *Archiv f. Ophthalmologie*, cv., p. 851.

179. HARBRIDGE, D. F.: **Sympathetic iridocylitis.** *American Journal of Ophthalmology*, October, 1922.

180. HIRSCH. **Is there such a thing as sympathetic ophthalmia?** *Deutsche med. Wochenschrift*, 1921, p. 158.

181. JAKOBI. **A case of acquired syphilitic panophthalmitis with the histological condition.** *Zeitschrift f. Augenheilkunde*, xlvi., p. 133.

182. JELLINEK, ST. **Monolateral immobility of the pupil and Horner's syndrome after electric trauma.** *Ibid.*, xlvi., 1921.

183. KAFKA. **A peculiar pupillary condition.** *Archiv f. Ophthalmologie*, cv., 1921.

184. KLEINSASSER, E. **Disturbances of the visual field in iridocyclitis.** *Klin. Monatsbl. f. Augenheilkunde*, lxvii., 1921.

185. RAD. **Bilateral reflex immobility of the pupils after trauma to the skull.** *Deutsche Zeitschrift f. Nervenheilkunde*, lxviii.-lxix., 1921.

BEHR (171, **Abduction phenomenon of the pupil**) says that in many cases of absolute immobility of the pupil, less often in reflex immobility, a pathological movement of the sphincter pupillæ can be observed in association with abduction. As a rule the contraction of the pupil is only on one side, and it occurs only when the impulse to abduction is abnormally strong. The reaction does not begin coincidently with the forced abduction, but starts somewhat later. But the redilatation of the pupil begins immediately with the cessation of the innervation to abduct. In all of these cases the reaction

of the pupils to closure of the lids is abnormally distinct. The cause seems to be located in the central supranuclear tract.

BLIEDUNG (172, **Spontaneous intraepithelial cyst of the iris**) reports the case of a woman 53 years old who was operated on for bilateral cataract, and presented many brown swellings at the edge of the pupil, which were apparently cystlike dilatations of the pigment layer. During the operation for cataract on the right eye one of these cysts was seized and ruptured, when all collapsed. Little floating shreds remained at the margins of the incision. Recurrence after eleven months. The cysts burst likewise during the operation of the left eye; no recurrence has been observed. Histological examination of a piece of the cyst excised in the iridectomy showed its origin from the pigment epithelium.

DREYFUSS (173, **Prognostic guides in isolated syphilitic disturbances of the pupils**) says that anomalies in the size and form of the pupils occur in healthy persons, hence unequal and distorted pupils can be looked upon only as suspicious, but disturbances of the reactions to light and convergence are always pathological. When such other causes have been excluded as local disease of the eye, injuries of the eyeball and orbit, anteriosclerosis in the brain, injuries of the skull, morphinism and other chronic poisonings, diabetes, and epidemic encephalitis, an isolated pupillary disturbance is most probably syphilitic. Dreyfuss has observed for a long time 107 persons having positively syphilitic pupillary troubles, and comes to the following conclusions. The kind of pupillary disturbance gives us no clue as to the later fate of the patient. At any rate one cannot say positively that a patient with marked reflex immobility is now on the way to tabes. The slightest anisocoria with slowed reaction to light may be a precursor of paralysis. Reflex or absolute immobility is under certain circumstances only "the visiting card of a cured syphilis." Prognostic conclusions may be drawn from the Wassermann reaction of the cerebrospinal fluid. The writer divides his material into two main groups, liquor positive and liquor negative, with the subgrouping of serum positive and serum negative according to the Wassermann findings in the blood. Almost a third of the entire number of the liquor positive cases were serum negative, a proof of the unreliability

of the blood examination in nerve syphilis. Fifty-eight of the seventy-one liquor positive cases, 81%, had pupillary disturbances and symptoms pointing to an organic disease of the central nervous system. The difference in age between the liquor positive and liquor negative groups was striking; more than half of the former were between 30 and 39 years of age with extremely few over 45. This can mean only that the liquor positives die early. Another interesting point is that one-half of the patients claimed that they were not aware they had syphilis. If we assume that twenty per cent. made false statements there still remain thirty per cent., almost a third of all patients, who did not know of their infection, had never noted primary or secondary symptoms. If this is correct every third victim of nerve syphilis knows nothing of his infection. As regards the interval between the infection and the onset of the pupillary symptoms, it was from six to ten years in the majority, in no case over thirty years. Again the explanation is advanced that most of the patients die early. In the liquor negatives there was only half as large a percentage of pupillary changes, and in a quarter of these the pupillary troubles appeared more than thirty years after infection. More than two thirds of the liquor positives needed hospital care within the first fifteen years, usually on account of nervous troubles. Signs of aortitis were present in about 33% of both liquor positives and liquor negatives. Scarcely a patient complained of heart trouble at any time, the condition was found objectively. Twelve of the thirty-seven patients with aortitis were serum negative. A liquor positive patient with isolated syphilitic pupillary disturbance is suffering from an active, more or less rapidly progressive syphilis of the brain which may develop into a cerebrospinal syphilis, a tabes, or a general paresis. In liquor positive cases treatment cannot always check the disease and the prognosis is not good. In liquor negative cases the cerebral syphilis can probably be brought to a standstill.

GUIST (177, **Behavior of light and dark irides to homatropine and atropine**) has observed that homatropine is inefficient as a cycloplegic in some eyes, and that in them atropine requires more time to produce a complete paralysis, and has sought to determine the peculiarities whereby an iris which will

react well to these drugs may be differentiated from one which will not. He finds that light and dark irides react differently, and that this is the case also when a person has one light and one dark iris. Abundance of crypts, such as is found in light irides, favor the absorption of the drops, while a poverty of these in the dark irides furnish an obstruction to absorption. His conclusions are: 1. All light irides have crypts and may be paralyzed by homatropine. 2. Some dark irides have crypts, some have few or none; those with crypts can be paralyzed by homatropine the same as the light ones when the relief is delicate, but only partially when the relief is compact. 3. Brown irides without crypts cannot be influenced by homatropine and can be paralyzed only by atropine. The reason seems to be the thickness of the anterior limiting layer and the density of the iris stroma.

JELLINEK (182, **Monolateral immobility of the pupil and Horner's syndrome after electric trauma**) reports the case of a man 32 years old who was not killed by a current of 5000 volts. He was not rendered senseless, but suffered only a slight shock with transient confusion and recovered within a few hours. The burns healed smoothly and no changes appeared in his internal organs or nerves. But two hours after the accident the left eye was enophthalmic with a narrow palpebral fissure and a pupil scarcely as large as a pinhead. The pupil was immobile to light and convergence. No injury to the eye or its surroundings was visible. Twenty-four hours later the left pupil was still totally immobile; three days later it was smaller than the right but reacting to light and convergence. At the end of three weeks the Horner's syndrome had completely disappeared and the two eyes looked alike.

KAFKA (183, **A peculiar pupillary condition**) reports the case of a syphilitic man 35 years old in whom the following points were noted during the retrogression of an ophthalmoplegia externa of the right eye. When looking at a distance with moderate illumination the right pupil was considerably smaller than that of the normal left eye. While the man continued to look in the distance the right pupil gradually grew larger until at the end of twenty seconds it was considerably larger than the left. The right pupil was immobile to light, the left reacted

normally. Both pupils reacted to convergence and accommodation, the right very quickly and strongly, the left normally. Consensual reaction to light was present in the left eye, but not in the right. This condition of the pupil disappeared with recovery from the ophthalmoplegia. Kafka thinks that this was an isolated reflex pupillary immobility located in the centrifugal reflex arc, and that its occurrence makes probable the existence of special fibers for the convergence and light reflex in the oculomotor trunk. It is also suggested that the existence of inhibitive fibers for the sphincter in the oculomotor nerve is not improbable.

Frequently the ophthalmoscope fails to reveal why the vision is poor in iridocyclitis, while the perimeter may show central, paracentral, or peripheral scotomata which sometimes pass away later at the time when the vision improves. Kleinsasser (184, **Disturbances of the visual field in iridocyclitis**) present Meller's histological studies which go to show that material may wander from the diseased ciliary body along the retinal vessels behind the lamina, in the form of nodules of epithelioid cells found exclusively in the perivascular lymph spaces about the retinal veins. The parenchyma of the retina itself is free and the choroid is scarcely involved in the pathological process. The exciter of disease may also be carried from the uvea to the retrobulbar portion of the optic nerve through a periphlebitis of the retinal vessels.

Rad (185, **Bilateral reflex immobility of the pupils after trauma to the skull**) reports a case of this nature observed after a fracture of the base of the skull in a man 34 years old. Syphilis was excluded. The cause was believed to be a hemorrhage in the midbrain where the centripetal pupillary fibers split up about the nucleus of the sphincter.

Gilbert (176, **Benign tuberculous meningitis in choroiditis**) calls attention to two distinct forms of tuberculous choroiditis. One is miliary tuberculosis, the foci of which are often situated so far peripherally that they cannot be observed with the ophthalmoscope. The other form is that of disseminated choroiditis, which is frequently seen in persons apparently healthy and is caused by syphilis or tuberculosis. Just as the choroid provides nutriment for the retina, so the meninges provide for the brain, and these are often affected by a benign

meningitis. In these cases tuberculosis of the bronchial glands is present, and the choroiditis is associated with headaches which last weeks or months, and an increased lumbar pressure, that are not to be explained through the choroidal disease. These symptoms indicate meningeal trouble. This headache is present in the beginning of an attack of choroiditis in about eighty per cent. of the cases.

In HANKE'S (178, **Septic metastases in the choroid**) case it was possible to observe the changes within the eye because the vitreous remained clear. In one eye, besides hemorrhages which formed about the papilla and extended toward the macula, there were three egg shaped, sharply defined spots, two of which had red centers while the third had a white center. Three similar white spots were present in the other eye. Microscopic examination showed that the metastasis was not in the retina, but started from the choroid. In one circumscribed area of the choroid the stasis was so great that the sheaths of the vessels disappeared. There was then stasis and thrombosis, likewise leucocytes, and a small celled infiltration developed. In places the vessels ruptured so that blood accumulated beneath the retina, in other places a septic focus could be seen in the choroid over which the retina was on the whole little changed; the pigment epithelium covering it was destroyed and a layer of pus was spread over the hyaloid membrane. In corresponding places were retinal hemorrhages, surrounded here and there by mantles of round cells. In some places the hyaloid membrane was broken through so that the focus in the choroid blended with the subretinal. On each side of such a focus a gaping vessel is often to be seen, which may become thrombosed later in the course. It is therefore supposed that the focus arises from an occlusion of a vessel. The subretinal focus may sink so as to be situated deeper than the one in the choroid and appear to have no connection with it.

JAKOBI (181, **Acquired syphilitic panophthalmitis**) examined the eye of a man 47 years old who had acquired syphilis four months before. The cornea was diffusely opaque, with deposits and a hypopyon. The iris was overfilled with blood, the tension increased. The tension continued to rise, the eye became blind and the eyeball was removed at the end of

three weeks. The other eye presented iritis, retinal hemorrhages, hazy papilla, narrow and irregular arteries, and some choroidal patches. Examination of the enucleated eye revealed perivasculitis in all sections. In the cornea there was a new formation of vessels with few cells, while the superficial vessels of the anterior portion of the sclera had many cells. The anterior chamber contained red and white corpuscles. The iris was uniformly permeated. The vessels showed sclerosis and hyaline degeneration with irregularities of the endothelium. The cells were chiefly lymphocytes with a few plasma cells. The ciliary body was permeated chiefly with plasma cells, the pigment epithelium irregularly degenerated. The choroid presented exudates, chiefly of lymphocytes and plasma cells, most marked anteriorly and at the optic nerve. The retina showed prominent signs of degeneration and the vessels were changed. A uniform infiltration occupied the optic nerve and its sheath, the nerve fibers were destroyed. At the posterior pole of the eye was a dense felt of tissue with vessels. Aside from fibroblasts lymphocytes and plasma cells could be found, and in some places there was a tissue poor in nuclei. The sclera also was infiltrated with cells. Jakobi thinks there had been an irruption of spirochætæ into the posterior ciliary arteries. All parts of the eye were affected, but the choroid to the greatest extent. Tuberculosis was contraindicated by the absence of reaction as well as by the nature of the occlusion of the arteries, which was in this case through a perivasculitis instead of thrombosis as is usual in tuberculosis. Yet the Wassermann was negative.

Bartok (170, **Sympathetic ophthalmia of long duration**) has observed several cases of sympathetic inflammation which ran a benign course for from eighteen to twenty-three years. The vision was fair for a long time. One eye was myopic. An affected eye is never to be trusted, even when quiet. The plastic forms tend soonest to recurrence. As long as the first eye has vision it should not be removed as the course is often uncertain and frequently this eye will eventually have the better vision.

In an eye removed at the beginning of a sympathetic inflammation Fuchs (174, **Sympathetic inflammation and sarcoma in the same eye**) found a small melanosarcoma of the choroid

near the equator. In addition to this there was in the same eye an adenoma in a ciliary process.

FUESTI-MOLNAR'S (175, **Sympathetic ophthalmia after trephining**) case was one in which trephining had been done for absolute glaucoma of one eye, the other being normal. No reduction of tension followed the operation. Five days later ciliary redness appeared and iritis developed. The tension fell considerably and then the inflammation subsided so that the patient was discharged. Seven weeks after the operation the healthy eye developed a severe iridocyclitis, its vision falling to $\frac{5}{10}$, while the eye that had been operated on showed the same inflammatory symptoms. Four days later the first eye was removed, after which the symptoms of inflammation in the second rapidly subsided with a return of nearly normal vision. The trephine opening was found to be filled with granulation tissue containing leucocytes, lymphocytes, and pigment, together with infiltrated iris and with the lens adherent. The capsule of the lens had been wounded so the lens fibers were swollen.

HARBRIDGE (179, **Sympathetic iridocyclitis**) discusses the various theories as to the cause of this disease and cites a case of sympathetic ophthalmia in which the teeth were badly infected. He thinks that the injury to the sympathogenic eye brings about a condition which renders it susceptible to endogenous infection of focal origin. The second eye he believes is sensitized as an anaphylactic phenomenon.

HIRSCH (180, **Is there such a thing as sympathetic ophthalmia?**) answers his question in the negative and explains the cases as tuberculous. He claims that no proof exists of the dependence of the inflammation of the second eye upon the injury to the first, and that the inflammation is as spontaneous in the one eye as in the other. All of the cases he has seen were in tuberculous patients, and he has found treatment with tuberculin to be beneficial. The beneficial effect of enucleation he explains as from a venesection, which causes an increase in the content of antitoxin, like that produced by such other remedies as mercury. Also in tuberculosis intervals of many years can be seen, so a reactivation is always possible.

XII.—THE RETINA AND OPTIC TRACT.

186. BEHR. **The anatomy of the senile macula.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 551.

187. CASSIDY, W. A., and GIFFORD, S. R. **An unusual circular lesion of the retina.** *American Journal of Ophthalmology*, June, 1922.

188. DANCO. **Bilateral stellate central neuroretinitis after grippe.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 87.

189. FUCHS, E. **Chorioretinitis.** *Ibid.*, August–September, 1921, p. 326.

190. GARVEY, J. L. **Hysterical homonymous hemianopsia.** *American Journal of Ophthalmology*, September, 1922.

191. GOURFEIN-WELT. **Retinitis caused by ethmoiditis.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 332.

192. GROENHOLM. **Preëquatorial sclerectomy in detachment of the retina.** *Archiv f. Ophthalmologie*, cv., 1921.

193. IGERSHEIMER. **Spirochætæ findings in the optic tract in general paralysis.** *Deutsche med. Wochenschrift*, 1921, No. 26.

194. KRAUPA and HAHN. **Spasmodic ischæmia of the inner vessels of the eye as a symptom of hereditary syphilitic angiopathy.** *Klin. Monatsbl. f. Augenheilkunde*, June, 1921.

195. LUTZ. **A rare form of disturbance in the visual field.** *Ibid.*, May, 1921.

196. MAERTENS. **A primary malignant epithelial tumor within the eye of an adult.** *Archiv f. Augenheilkunde*, lxxxix., p. 1.

197. MOORE, FOSTER. **An unusual case of renal retinitis.** *British Journal of Ophthalmology*, May, 1922.

198. RAIA, V. L. **Two cases of retinitis proliferans of syphilitic and diabetic origin.** *American Journal of Ophthalmology*, December, 1922.

199. SCHIOETZ, I. **Retinitis gravidarum and amaurosis eclamptica.** *Klin. Monatsbl. f. Augenheilkunde*, December, 1921.

200. SIDLER-HUGUENIN. **Retinal change in a case of chloroma.** *Ibid.*, July, 1921, p. 55.

201. WAGENER, H. P. **Lipæmia retinalis.** Reports of three cases. *American Journal of Ophthalmology*, July, 1922.

202. WUERDEMANN, H. V. **Retinitis proliferans.** *Ibid.*, May, 1922.

BEHR (186, **Anatomy of the senile macula**) examined a case of senile degeneration of the macula and found true degenerative changes without reactive peculiarities. The sensory epithelium in the foveola was the most affected, and from this point the destruction of tissue spread uniformly in graduated steps. There were no changes of the pigment epithelium or of the choriocapillaris.

MOORE (197, **An unusual case of renal retinitis**) gives an account of a case of renal retinitis which was under observation for 7½ years. This case, a male aged 45 years, first

attended hospital for failing vision. The retinal changes were marked, a detachment of the retina being present in each eye. The blood pressure was 215*mm*, the heart was hypertrophied and the urine loaded with albumen.

After 8 weeks treatment the detachments in each eye had disappeared.

After 5 months there was marked improvement in the condition of each retina.

After 19 months the only change in either fundus were signs of secondary atrophy of the disks, attenuation of the vessels and a little exudate. His blood pressure was now 190*mm* and there was no albumen in the urine.

He was not seen again until 7 years later. He stated that he was in good health and had been so since his last visit to hospital. On examination of the fundi there were no exudates or hemorrhages to be seen, secondary atrophy of the disk and attenuation of the vessels were the only signs present.

Shortly after this visit he was taken suddenly ill and in a few weeks died in uræmia.

This case is of interest in the unusually long survival of life after the onset of the renal retinitis and further in the subsidence of the retinitis with no recrudescence of it in the final stages of the disease.

Foster Moore thinks that it is questionable whether a retinitis, once subsided, can recur and suggests that an eye which has been the seat of serious pathological disturbance may acquire a protection against any toxæmia which in the ordinary course of events would produce a retinitis.

The post-mortem of the above case, which was conducted by Dr. Geoffrey Evans, showed a condition of chronic mixed nephritis and arteriosclerosis. The eyeballs were excised and examined by Mr. Humphrey Neame who reported that there was no sign of detachment in either eye, that the retinal vessels were very thick walled, and that there was no sign of exudate in the retina with this exception that in the left eye there was a slight amount of exudate in Henle's layer near to the disk together with a peculiar localized pigment proliferation in the same region.

CASSIDY and GIFFORD (187, **Unusual circular lesion of the retina**) describe a lesion of the fundus which showed two

concentric incomplete white rings about the macula with a large central relative scotoma. They appeared to be slightly elevated. A week later the rings had nearly disappeared. As the patient had received many blows on the eye it was thought that there may have been a slight detachment. His Wassermann reaction however was + + . Only one other similar case was found in literature.

RAIA (198, **Retinitis proliferans of syphilitic and diabetic origin**) had the opportunity to examine his patients for long periods and at no time observed any hemorrhages except a small spot on the surface of the new formed membrane, in one case and a few in the retina of the other. This fact seems to controvert the opinion that retinitis proliferans is preceded by hemorrhages.

WUERDEMANN (202, **Retinitis proliferans**) reports two cases which are of interest because of the excessive development of varices and newly formed vessels on the optic disks.

DANCO (188, **Bilateral stellate central neuroretinitis after grippe**) observed a case of this nature for sixteen months, during which time the condition underwent considerable changes. On one side the papilla became much paler, on the other there was a partial retrogression of the stellate figure and patches formed in the periphery of the retina. Associated with the more typical stellate figure in the macula there were diffuse patches surrounding at some distance the nasal, upper and lower margins of the papilla. These patches later became little white spots. Pigment spots also appeared later in the affected parts of the retina. The vision at first was right 1.$\frac{5}{5}$ left fingers at 50*cm*, but finally improved to right $\frac{5}{5}$, left $\frac{2}{5}$. The color sense was even more greatly disturbed.

FUCHS (189, **Chorioretinitis**) raises the question which membrane is primarily affected in chorioretinitis. In a case of old chorioretinitis he found purely retinal foci, while the pigment epithelium and choroid were intact at these places. It is assumed that in this case the starting point of the inflammation was in the retina, but Fuchs believes that in most cases the starting point is in the choroid.

GOURFEIN-WELT (191, **Retinitis caused by ethmoiditis**) saw a case of retinitis in a woman suffering from purulent ethmoiditis. The macula was dark red and surrounded by a glisten-

ing wreath of white lines. Vision promptly improved as soon as the ethmoid cells had been cleaned out.

SCHIOETZ (199, **Retinitis gravidarum and amaurosis eclamptica**) examined ophthalmoscopically 680 out of a total of 8400 pregnant and lying-in women. In forty cases retinitis of pregnancy was diagnosed. Twenty-seven showed signs of chronic renal trouble, ten of whom probably had nephritis before the beginning of pregnancy. Six patients showed no signs of kidney trouble. No subsequent examination was made of seven. Retrogression takes only a few days or weeks after delivery, the changes disappear leaving scarcely any trace. Retinal hemorrhages were common, but did not indicate a bad prognosis as to life. The retinal changes increase up to the time of delivery, and in general the prognosis as to sight depends on the time of the interruption of the pregnancy. Among 132 patients with eclampsia and 26 threatened with the same he found 27 cases of retinitis, four of them complicated by detachment of the retina. Twenty of the patients examined showed no eye symptoms. In his description of eclamptic amaurosis he mentions one patient who suffered from transient hemianopsia after the blindness passed off, and two who had disturbances of the color sense. Four had choked disks. There were also four cases of choked disk in cases of eclampsia without amaurosis.

SIDLER-HUGUENIN (200, **Retinal change in a case of chloroma**) saw a peculiar case of chloroma which began suddenly with bleeding from the gums and proved fatal within three weeks. Striated and lakelike hemorrhages were formed in the retina, resembling those characteristic of leukæmia. They were situated for the most part in the nerve fiber, ganglion cell, and outer granular layers.

WAGENER (201, **Lipæmia retinalis**) describes the characteristic picture of this disease as showing an optic disk, waxy in appearance, narrow retinal arteries, pinkish color at the disk fading rapidly as the vessels divide. The veins on the disk are deep red fading toward the periphery. In the cases previously reported lipæmia giving rise to retinitis has been incident to the course of a severe diabetes mellitus and appears only when the fat content of the blood goes above 4%. Seventeen cases of this disease have appeared in literature.

KRAUPA and HAHN (194, **Spasmodic ischæmia of the inner vessels of the eye as a symptom of hereditary syphilitic angiopathy**) saw a peculiar case of embolism of the central artery in a girl 5¾ years old. At the first examination the retinæ was white as silver with red spots in the maculæ which looked like hemorrhages. The veins were much dilated, the arteries of about normal size with uninterrupted circulation. Some days later the papillæ were pale, the retinal opacity less, vision of the right eye perception of light, of the left counting fingers. He diagnosed a spasm of the capillaries and prescribed papaverin. Under this treatment the condition improved, the papillæ again became pink and vision rose to right, 0.5, left 1.0.

GARVEY (190, **Hysterical homonymous hemianopsia**) reports the case of a young woman, 26 years of age, who developed left homonymous hemianopsia after an attack of pain, dizziness and vomiting. It persisted for a number of months. She gave the history of a severe blow over the right eye two months before, but that was thought to have no relation to the hemianopsia. The questionable diagnosis of endarteritis of the retinal vessels was made. As she was hysterical and recovered later, the phenomenon was ascribed to this cause.

GROENHOLM (192, **Preëquatorial sclerectomy in detachment of the retina**) has performed this operation in forty-one cases. Four were cured, seventeen improved. When the final result is taken into consideration a transient effect is found in the majority of cases. In ten per cent. the detachment disappeared, in twenty-four per cent. it was unchanged, and in sixty-seven per cent. it was enlarged. The size of the detachment is important, the smaller it is the better the result. The earlier the operation is performed the better also.

IGERSHEIMER (193, **Spirochætæ findings in the optic tract in general paralysis**) has found spirochætæ in the optic tract in two cases of progressive paralysis, one with a beginning and one with an advanced optic atrophy, in one case just behind the chiasm and in the corpus geniculatum, in the other in sections of the chiasm.

LUTZ (195, **Rare disturbance in the visual field**) reports the case of a patient who was supposed to have a tumor of the brain in the middle fossa after traumatism. A simple gray optic atrophy developed in each eye, and spots resembling

albuminuric retinitis were present for a time. The sense of smell was completely lost on the left side and diminished on the right. The visual field showed heteronymous quadrant defects, on the right side above, on the left below, binasal tetranopsia. Finally it became very much contracted on the right side below the horizontal plane, on the left side above, heteronymous horizontal hemianopsia. The supposition was advanced that both optic nerves were constricted by the anterior cerebral artery just in front of the chiasm.

MAERTENS (196, **Primary malignant epithelial tumor within the eye of an adult**) found in an eye which had been small and blind from youth and enucleated because of increased tension from a woman 46 years old, a tumor that extended within the optic nerve to the base of the brain. On autopsy there was found an extensive growth in the region of the chiasm which had pushed the other optic nerve to one side and flattened it. As no other tumor was found elsewhere in the body this one had to be regarded as starting primarily from the eye.

XIII.—TRAUMATISMS.

203. HILDESHEIMER. **Wandering of an intraocular foreign body.** *Klin. Monatsbl. f. Augenheilkunde,* June, 1921, p. 931.

HILDESHEIMER (203, **Wandering of an intraocular foreign body**) reports the following interesting case. In 1907 the eye of a man 30 years was wounded by a splinter of iron. Several attempts to remove the foreign body failed. Eight years later the patient again came under observation because of an iritis; there was a scar at the limbus and the sideroscope strongly indicated the presence of a foreign body. There was no siderosis, the tension was low, there was a circle of posterior synechiæ, and vision was reduced to movements of the hand. In 1920, thirteen years after the accident, the patient returned again because of pain in the eye. The point of the splinter appeared in the scar so that finally it could be withdrawn with forceps. The remains of the lens became absorbed and vision became $\frac{5}{15}$ with cataract glasses.

(*Concluded.*)